University of Kentucky
UKnowledge

Theses and Dissertations--Plant and Soil Sciences

Plant and Soil Sciences

2019

IMPACT OF A WARMED ENVIRONMENT, SPIKE MORPHOLOGY AND GENOTYPE ON FHB LEVELS IN A SOFT RED WINTER WHEAT MAPPING POPULATION

Elisane Weber Tessmann
University of Kentucky, lisa.tessmann@uky.edu
Author ORCID Identifier:
https://orcid.org/0000-0002-1517-9627
Digital Object Identifier: https://doi.org/10.13023/etd.2019.150

Right click to open a feedback form in a new tab to let us know how this document benefits you.

Recommended Citation

Weber Tessmann, Elisane, "IMPACT OF A WARMED ENVIRONMENT, SPIKE MORPHOLOGY AND GENOTYPE ON FHB LEVELS IN A SOFT RED WINTER WHEAT MAPPING POPULATION" (2019). *Theses and Dissertations--Plant and Soil Sciences*. 116.
https://uknowledge.uky.edu/pss_etds/116

This Doctoral Dissertation is brought to you for free and open access by the Plant and Soil Sciences at UKnowledge. It has been accepted for inclusion in Theses and Dissertations--Plant and Soil Sciences by an authorized administrator of UKnowledge. For more information, please contact UKnowledge@lsv.uky.edu.

STUDENT AGREEMENT:

I represent that my thesis or dissertation and abstract are my original work. Proper attribution has been given to all outside sources. I understand that I am solely responsible for obtaining any needed copyright permissions. I have obtained needed written permission statement(s) from the owner(s) of each third-party copyrighted matter to be included in my work, allowing electronic distribution (if such use is not permitted by the fair use doctrine) which will be submitted to UKnowledge as Additional File.

I hereby grant to The University of Kentucky and its agents the irrevocable, non-exclusive, and royalty-free license to archive and make accessible my work in whole or in part in all forms of media, now or hereafter known. I agree that the document mentioned above may be made available immediately for worldwide access unless an embargo applies.

I retain all other ownership rights to the copyright of my work. I also retain the right to use in future works (such as articles or books) all or part of my work. I understand that I am free to register the copyright to my work.

REVIEW, APPROVAL AND ACCEPTANCE

The document mentioned above has been reviewed and accepted by the student's advisor, on behalf of the advisory committee, and by the Director of Graduate Studies (DGS), on behalf of the program; we verify that this is the final, approved version of the student's thesis including all changes required by the advisory committee. The undersigned agree to abide by the statements above.

Elisane Weber Tessmann, Student

Dr. David Van Sanford, Major Professor

Dr. Mark S. Coyne, Director of Graduate Studies

IMPACT OF A WARMED ENVIRONMENT, SPIKE MORPHOLOGY AND GENOTYPE ON FHB LEVELS IN A SOFT RED WINTER WHEAT MAPPING POPULATION

DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy in
the College of Agriculture, Food and Environment
at the University of Kentucky

By
Elisane Weber Tessmann

Lexington, KY

Director: Dr. David Van Sanford, Professor of Crop Science

Lexington, Kentucky

2019

Copyright © Elisane Weber Tessmann 2019
https://orcid.org/0000-0002-1517-9627

ABSTRACT OF DISSERTATION

IMPACT OF A WARMED ENVIRONMENT, SPIKE MORPHOLOGY AND GENOTYPE ON FHB LEVELS IN A SOFT RED WINTER WHEAT MAPPING POPULATION

Fusarium head blight (FHB) is a serious disease of wheat (*Triticum aestivum*) and other small grains; disease severity is affected by temperature and rainfall. This research comprised three studies: an artificially warmed experiment during 2016-2017, a morphology study and an FHB resistance screening study in 2015-2016, using approximately 250 wheat cultivars and breeding lines from programs in the eastern US. The location was the University of Kentucky Spindletop Research Farm in Lexington, KY. Higher levels of Fusarium damaged kernels and the toxin deoxynivalenol (DON) were observed in the warmed treatment compared to the control, and plant development was accelerated. In the FHB resistance screen, significant ($p<0.05$) genotype differences for all traits were observed. A GWAS identified 16 SNPs associated with resistance and susceptibility, ranging from -2.14 to 4.01%. Three DON-associated SNPs reduced toxin levels by 3.2, 2.1, and 1.5 ppm. In the morphology study, negative correlations were observed among morphological and disease traits. Small effect SNPs were identified for all morphological traits, which might be useful in genomic selection; traits like spike length, spikelet number and inclination could be used in phenotyping. Response to warming indicates that existing resistance sources may be less effective in a warming climate.

KEYWORDS: Artificially warmed environment, spike morphology, scab traits, GWAS, SNP

Elisane Weber Tessmann

04/25/19

Date

IMPACT OF A WARMED ENVIRONMENT, SPIKE MORPHOLOGY AND GENOTYPE ON FHB LEVELS IN A SOFT RED WINTER WHEAT MAPPING POPULATION

By

Elisane Weber Tessmann

Dr. David Van Sanford
Director of Dissertation

Dr. Mark S. Coyne
Director of Graduate Studies

04/25/19

Date

ACKNOWLEDGMENTS

The conclusion of this dissertation would not be possible without the guidance and assistance of a number of individuals. First and foremost, thank you to my advisor Dr. David Van Sanford for his guidance, support, patience and constant encouragement throughout this dissertation. Under his leadership, I was able to grow personally and professionally, and I feel confident and prepared for the future. I will be forever grateful for the opportunity that he gave me. I would also like to thank the members of my committee, Dr. Tim Philips, Dr. Carl Bradley and Dr. Hongyan Zhu, for their contributions. I also thank Dr. Bruce Downie for his contribution as an outside examiner.

My special thank you to John Connelly, Sandy Swanson and Anthony Clark for their support during field experiments. In addition, thank you to past and present Wheat Breeding Program members and summer undergraduate students that helped the execution of this work. I would also like to thank Dr. Ezequiel de Oliveira and Dr. Daniela Sarti Dvorjak for helping me to come for a summer internship under Dr. David Van Sanford supervision, which led me to start the PhD program. I want to thank many graduate students for their friendship during these years, in special to Ezequiel de Oliveira, Andrea Sanchez, Katlyn Hitz, Kathleen Russell, Ethan Swiggart, Katherine McLachlan Rod, Virginia Verges and Jesse Carmack.

I am deeply thankful to my parents and sister for always been supportive during this journey and to my boyfriend Bryan McMorrow for being my partner supporting me in the good and bad moments.

Table of Contents

LIST OF TABLES

LIST OF FIGURES

LIST OF SUPPLEMENTAL TABLES

LIST OF SUPPLEMENTAL FIGURES

LITERATURE REVIEW

Introduction

Wheat (*Triticum aestivum* L.) is one of the most important cereal crops and it has been part of human civilization since its domestication around 10,000 years ago (Eckardt, 2010). This cereal accounted for more than 15% of the crop land harvested in the world with over 218 million hectares of farmland worldwide, followed by corn (*Zea mays*) and rice (*Oryza sativa*) in 2017 (Food and Agriculture Organization (FAO) 2019). In terms of production, wheat produced a little bit over 35,000 hg ha^{-1}, behind corn, rice and triticale (*Triticale hexaploide*) in 2017 (FAO, 2019).

The Food and Agriculture Organization recently published that with nearly 821 million of people, 1 out every 9, in 2017 were undernourished (FAO, 2018). The organization's target is by 2030 to end hunger and ensure that all people have access to food (FAO, 2018). Many factors are involved in food security; governmental conflicts, wars, climate conditions, agricultural practices, among others. Breeders and other agronomist face the challenge of increasing production with minimal or no increase in area. In addition, climate change with increases of extreme events, drought periods or excesses water, add an additional layer of difficulty in the production system (Tataw et al., 2016; Allen et al., 2018). Another important player is the occurrence of plant diseases where many factors can affect disease occurrence and intensity. For Fusarium head blight (FHB), one of the most important diseases in wheat, weather conditions are important. In this scenario, breeders are under increased pressure to develop new cultivars to keep up with the increasing food demand by producing more under variable climatic conditions.

Fusarium head blight

Fusarium head blight (FHB), also known as head scab, is a fungal disease caused by several *Fusarium* species. In the eastern half of the U.S., the primary agent is *Fusarium graminearum* Schwabe (syn. *Gibberella zeae* (Schwein.) Petch), but others such as *F. culmorum* and *F. avenaceum* can also cause the disease in cooler and humid conditions (Parry et al., 1995; He et al., 2016a). In wheat (*Triticum aestivum* L.) and other small grain crops, FHB is one of the most important diseases, in which reduced yields, low test weight, and mycotoxin contamination can affect the final price (Balut, 2012). Losses caused by FHB were estimated at $4.8 billion in United States during the 1990s (Johnson et al., 2003). In epidemic years of 1998 through 2000, $2.7 billion in direct and indirect losses affected wheat and barley (*Hordeum vulgare* L.) in US (Nganje et al., 2002). Other regions in the world also have experienced losses due to FHB infection, for example, in southern Brazil, yield losses ranged from 11.6 to 39.8% from 2000 to 2010 (Reis and Carmona, 2013).

FHB is strongly driven by weather conditions, where warm and wet environments are the perfect conditions for disease development (Vaughan et al., 2016). Management practices such as minimal or no tillage and rotation with host crops like corn can intensify disease occurrence and symptoms (Chakraborty and Newton, 2011; McMullen et al., 2012; Steiner et al., 2017). Crop residue from cultivation of corn, barley, soybeans (*Glycine max*) and dead tissue are alternative hosts for perithecia and serve as primary inoculum (Bai and Shaner, 2004; McMullen et al., 2012). Spores deposited in crop debris are dispersed through splashing and wind. The dominant form of inoculum is the ascospore produced from perithecia; conidia, however, can also cause infection (Vaughan et al., 2016). During

its saprophytic stage, the fungus produces deoxynivalenol (DON) as an antimicrobial metabolite that acts against other soil microorganisms (Audenaert et al., 2013).

Disease infection occurs during or just after anthesis, when open florets provide the opportunity for the pathogen to enter and initiate infection (Emrich et al., 2008). Infected florets may fail to produce grains, leading to a reduction in yield. In florets able to produce grain, shriveled kernels with light weight and discoloration, known as scabby kernels or tombstones, are observed (Bai and Shaner, 2004; Balut, 2012). FHB significantly reduced the percentage of high-molecular-weight glutenins and low-molecular-weight glutenins, two important components of gluten in a study reported by Spanic et al. (2017). During the infection stage, DON is produced and helps the pathogen spread through the rachis (Zhang et al., 2012). As a mechanism of defense, plants produce H_2O_2 to kill attacked cells (Audenaert et al., 2013). Some studies demonstrate that the production of H_2O_2 actually helps DON production by the pathogen which enables further spreading of disease (Audenaert et al., 2010).

Mycotoxins are characteristic of individual strains of the fungus; for *F. graminearum* the main chemotypes are type B trichothecenes where acetylated DON, acetyldeoxynivalenol (3-ADON and 15-ADON) and nivalenol (NIV) are the most common (Desjardins, 2003; Audenaert et al., 2010). DON is a major concern associated with FHB, because this mycotoxin can cause chronic disease symptoms if consumed by human and animals. Symptoms such as diarrhea, vomiting, gastro-intestinal inflammation, necrosis of the intestinal tract, among others are characteristic of humans and animals ingesting DON (Audenaert et al., 2013). The advisory levels of DON recommended by Food and Drug Administration (FDA) are: 1 ppm on finished wheat products that may potentially be

consumed by humans; 5-10 ppm on grains and grain by-product for animal consumption, depending on species and the proportion in their diet (FDA, 2010).

Host resistance

Successful infection and colonization by *Fusarium* ssp. that cause FHB depend on factors such as time of infection, fungal pathogenicity, host susceptibility and environmental conditions (Kugler et al., 2013; Vaughan et al., 2016). Integrated management practices such as resistant cultivars and FHB-specific fungicide applications during flowering are the most efficient techniques to control FHB (Cowger et al., 2016). Farmers can make decisions as to when is the best moment for fungicide application based on the disease risk using the FHB Prediction Center (http://www.wheatscab.psu.edu). Techniques such as RNA interference (RNAi) for fungicide development is a promising approach for controlling FHB (Machado et al., 2017). RNAi is a gene silencing mechanism that involves small RNA molecules and can be highly specific. One approach to this technique, would be to use spray-induced gene silencing, which uses the same mechanism as RNAi, by applying long double-stranded RNA and small interfering RNA (Meister and Tuschl, 2004; Machado et al., 2017).

Resistant cultivars are one of the most important ways to control or reduce the effects of FHB. FHB is a quantitative disease, involving multiple genes with major and minor effects and it may vary in different genetic backgrounds (Petersen et al., 2016). Mesterhazy et al. (1999) summarized five types of host resistance against FHB: i) resistance to initial infection (Type I); ii) resistance to spread of the pathogen inside the

plant (Type II); iii) resistance to toxin accumulation (Type III); resistance to kernel infection (Type IV); and tolerance (Type V).

The Chinese cultivar 'Sumai-3' probably is one of the most used sources of FHB resistance, where quantitative trait locus (QTL) *Fhb1* (chromosome 3BS), *Fhb2* (chromosome 5A), and *Qfhs.ifa-5A* (chromosome 6BS) were found (Buerstmayr et al., 2002, 2003, 2010; Kazan and Gardiner, 2017). The mechanism of *Fhb1* is to provide resistance against spread of disease (Type II resistance), while *Qfhs.ifa-5A* provides initial resistance to penetration (Type I resistance) (Buerstmayr et al., 2002, 2003). *Fhb4* and *Fhb5* are also sources of resistance found in the cultivar 'Wangshiubai' (Xue et al., 2010, 2011). Jiang et al. (2007a, b) identified another source of resistance, *Qfhs.nau-2DL* in the breeding line 'CJ 9306'. Bai and Shaner (2004) mentioned the Japanese cultivars 'Shinchunaga', 'Nobeokabouzu', and 'Nyu Bai' as having high levels of resistance and noted that the cultivar 'Shinchunaga' was successfully used in breeding programs. In Latin America, the cultivar 'Frontana' from Brazil, is considered to have moderate type I FHB resistance (Steiner et al., 2004; Buerstmayr et al., 2009).

Islam et al. (2016) summarized some of the QTL that have been mapped in European winter wheat cultivars: 'Arina' (Paillard et al., 2004; Draeger et al., 2007), 'Fundulea 201R' (Shen et al., 2003), 'Renan' (Gervais et al., 2003), 'Remus' (Steiner et al., 2004) and 'NK93604' (Semagn et al., 2007). In addition, Islam et al. (2016), mention several sources of resistance in U.S.: 'Freedom' (Gooding et al., 1997), 'Goldfield' (Ohm et al., 2000), 'Roane', 'McCormick', 'Tribute' and 'Jamestown' (Griffey et al., 2001, 2005a, 2005b, 2010), 'Ernie', 'Truman', and 'Bess' (McKendry et al., 1995, 2005, 2007).

Breeders around the world are working hard to understand FHB and develop resistant cultivars. Thanks to advances in molecular genetics a lot of progress has been made in breeding to reduce the incidence and/or severity of this disease. Jia et al. (2017) reviewing the literature found more than 250 QTL for FHB resistance on all 21 wheat chromosomes. In 2016, a map-based cloning of *Fhb1* was published, opening the possibilities for direct cloning of this gene (Rawat et al., 2016). In addition, the International Wheat Genome Sequencing Consortium (IWGSC, 2018) recently divulged the annotated reference genome for all 21 bread wheat chromosomes. With the sequence information, new techniques, such as genome editing, can be applied to accelerate breeding for improved agronomic and disease traits.

Morphological traits

Plant morphological traits play an important role by providing passive disease resistance or susceptibility to FHB (Steiner et al., 2017; Jones et al., 2018). As described by Jones et al. (2018), avoidance or escape does not depend on disease resistance genes; it is purely based on morphological traits, where it reduces the likelihood of contact between pathogen and plant even though both are present at the same time.

Morphological traits such as plant height, flowering time and duration, absence or presence of awns, anther extrusion, spike density, spikelet number can influence resistance to *Fusarium* spp. (Liu et al., 2007; Graham and Browne, 2009; Suzuki et al., 2012; Liu et al., 2013; Buerstmayr and Buerstmayr, 2016).

Plant height

Several studies associated plant height with resistance to FHB (Hilton et al., 1999; Klahr et al., 2007; Srinivasachary et al., 2008, 2009; Mao et al., 2010; Yan et al., 2011; Kollers et al., 2013; Lu et al., 2011, 2013; Buerstmayr and Buerstmayr, 2016; Schulthess et al., 2018). Three mechanisms were reported to be associated with plant height: disease escape, pleiotropy or tight linkage (He et al., 2016b). Yan et al. (2011) suggested that disease avoidance was due to physical distance between plant and inoculum, being due to plant height *per se*. When the authors physically raised semi-dwarfing genotypes to match wild type genotypes, differences in disease between genotypes were eliminated. A negative correlation between plant height and FHB was found by Klahr et al. (2007); they attribute the susceptibility of short genotypes to two factors: i) the spike's proximity to the inoculum, and ii) the microenvironment with high moisture around the spike. A pleotropic effect was demonstrated by Saville et al. (2012) studying the function of DELLA, a protein encoded by *Rht-B1b* and *Rht-D1b*. The authors showed the accumulation of DELLA by the semi-dwarf allele (*Rht-B1b*) and the severe dwarf allele (*Rht-B1c*) increased susceptibility to initial infection when compared with the wild type. In addition, they concluded that plants with gain in function of DELLA were more resistant to colonization and DON induced cell death probably due to reduced propensity to initiate cell death.

The semi-dwarfing alleles *Rht-B1b* and *Rht-D1b* are extensively used in breeding programs where both reduce plant height, however they show differences in terms of susceptibility to FHB (Steiner et al., 2017). Srinivasachary et al. (2009) demonstrated that the presence of *Rht-B1b* and *Rht-D1b* decreased resistance to initial infection, while the *Rht-B1b* increased resistance to spread of the pathogen inside the plant (Type II), *Rht-D1b*

had no effect on it. Other studies reported the association of *Rht-D1b* and decrease in resistance to initial infection (Draeger et al., 2007; Srinivasachary et al., 2008; Lu et al., 2011). Lu et al. (2013) indicated that for breeding the desirable dwarfing allele to be used in breeding is *Rht-B1b* due to its type II resistance.

Heading date

Heading date can impact FHB, since an early or late flowering period can provide escape from infection. Positive significant correlations between heading date and Fusarium damaged kernels (FDK) and DON were reported in the literature, indicating that early genotypes are more resistant to FHB (Liu et al., 2013; Petersen et al., 2016). However, climate change can potentially affect this correlation. In an artificially warmed experiment, 238 lines in a mist irrigation and scabby inoculum had an earlier heading date (3.5 days) and an increase of 84 and 131% was observed for DON and FDK, respectively (Tessmann and Van Sanford, 2018).

Heading date is controlled by vernalization response (*Vrn* genes), photoperiod sensitivity (*Ppd* genes) and earliness *per se* (*Eps* genes) (Herndl et al., 2008). Growth habit in wheat is determined by vernalization requirements, while in winter wheat high vernalization is required, in spring wheat low or no requirement is need to initiate flowering (Gomez et al., 2014). Three genes, *Vrn1*, *Vrn2* and *Vrn3* are responsible for regulating vernalization (Guedira et al., 2016). *Vrn1* is the most important one because it promotes the transition from vegetative to reproductive (Distelfeld et al., 2009; Trevaskis, 2010). In wheat, the homoeologous genes of *Vrn1* are *Vrn-A1*, *Vrn-B1*, and *Vrn-D1* on chromosomes 5A, 5B and 5D, respectively (Law et al., 1976; Herndl et al., 2008, Guedira et al., 2016).

The dominant allele in any genome confers spring growth, whereas, recessive alleles in the homozygous state confers winter wheat growth habit (Guedira et al., 2016). Diaz et al. (2012) demonstrated that the variation in vernalization requirement was due to the number of copies of *Vrn-A1*, where plants with more copies of *Vrn-A1* required longer periods of cold temperature.

Photoperiod genes are also related to the transition between vegetative and reproductive stage. Genotypes that rapidly flower when exposed to long days are described as photoperiod sensitive. Conversely, photoperiod insensitive genotypes flower in short or long days (Worland and Snape, 2001; Diaz et al., 2012, Guedira et al., 2016). Response to photoperiod is controlled by the *Ppd-1* gene, located on the short arm of chromosomes 2A, 2B and 2D (Gomez et al., 2014). The semi-dominant mutation *Ppd-D1a* was widely used in the "green revolution" because it provides photoperiod insensitive plants (Zanke et al., 2014). Grogan et al. (2016) observed that *Ppd-D1*, *Ppd-B1* and their interaction were responsible for most of the variation observed in heading date in 299 hard winter wheat genotypes. The allele *Ppd-A1* also affects photoperiod; however, its effect is weaker than *Ppd-D1* and *Ppd-B1* (Worland et al., 1998; Grogan et al., 2016). Earliness is described by Gomez et al. (2014) as being the variation observed in flowering after vernalization and photoperiod requirements were met. Griffiths et al. (2009) identified 19 meta-QTL for earliness in different regions in the chromosome.

Anther extrusion

Besides plant height and heading date, other traits are also reported to be associated with FHB. Anther extrusion during anthesis is observed in some cultivars, even though

wheat is considered an autogamous crop (Muqaddasi et al., 2017). De Vries (1971) described the factors controlling anther extrusion: turgidity of lodicule causes an elongation of the anther filaments which pushes apart lemma and palea. Low temperature and adequate humidity stimulate this mechanism, while high temperature and drought decrease it (Skines et al., 2010). Several studies reported an increase in FHB when anthers were retained in the florets (Graham and Browne, 2009; Skines et al., 2010; Lu et al., 2013; Buerstmayr and Buerstamayr, 2015). Skines et al. (2010) suggested that lines with trapped anthers between glumes were an easy target for colonization because they provided dead tissue. In addition, Kubo et al. (2013) demonstrated that closed-flowering or rapid anther extrusion lines were more resistant to FHB than lines where the anther was partially extruded. Gilsinger et al. (2005), studying F_2-derived recombinant inbred lines observed that plants with a wider flower opening had higher FHB incidence than those with narrow flower opening. Skines et al. (2010) identified QTL on chromosomes 1A, 1B, 4D and 6A that explained from 7.4 to 18.3% of the phenotypic variation for anther extrusion. A recent GWAS identified 23 marker trait associations, 11 in spring wheat and 12 in winter wheat for anther extrusion (Muqaddasi et al., 2017).

Spike morphology

Traits related to spike morphology, such as spike length, spikelet number, spike density and spike inclination could also be associated with passive disease resistance. A negative significant correlation between spike length and FHB was observed in some studies (Buerstmayr et al., 2011; Suzuki et al., 2012). For spikelet number, Liu et al. (2007) and Buerstmayr et al. (2011) did not find a significant correlation between spikelet number

and FHB, while Jones et al. (2018) found a positive correlation with DON. Spike density can influence the microclimate around the florets, where a dense spike (short spikes with high spikelet number) could increase humidity and favor fungal development (Jones et al., 2018). Positive significant correlations between spike density and FHB severity have been reported in the literature (Buerstmayr et al., 2011; Giancaspro et al., 2016, Yi et al., 2018). However, Steiner et al. (2004) observed different results where a negative correlation between spike density and FHB incidence was found for the cultivar Frontana. In barley, Urrea et al. (2002) found similar results, with a negative correlation between spike angle and FHB severity.

Morphological variation in certain spike traits can create a conducive environment for disease development (Jones et al., 2018). Thus, field evaluation and characterization of the population for spike traits can provide important information for breeders when selecting parents for crosses targeting FHB resistance.

Climate change

Increasing agricultural production in an environment that is constantly changing is one of the challenges of this century. Climate change, with rise in temperature and changes in rainfall patterns is estimated to affect global production of major crops by 6.0% in wheat, 7.4% in corn, 3.2% in rice and 3.1% soybean for each 1°C increased (Zhao et al., 2017). The Intergovernmental Panel on Climate Change (IPCC, 2014) projects a global increase in temperature of 1 to 3.7°C. However, decreases in crop production are being observed already; a study using data from 1964 to 2007 observed a decreased of 9-10% due to drought and extreme heat events (Lesk et al., 2016). Tack et al. (2015), studying Kansas

wheat varieties from 1985 to 2013, observed a decrease of 9% in yield due to days of freezing temperature in the fall, and 7.6% in yield in spring for each degree above 34°C. The authors indicated that the new varieties are less resistant to high temperature due to its longer grain-filling period prolonging plant exposure to environmental factors.

Greenhouse gas emission in the atmosphere increased levels of carbon dioxide (CO_2), methane (CH_4), and nitrous oxide (N_2O); around 60% of these gases are stored in plants and soil and thus not present in the atmosphere (IPCC, 2014). Constant release of CO_2 can increase temperature and cause fluctuation in precipitation, melting snow and ice which affects water resources (IPCC, 2014). Rosenzweig et al. (2014), using crop models observed a strong negative effect of climate change in crop production, indicating that plants are very sensitive to changes in the environment. However, the extent of damage depends on how quickly and to what degree temperature increases, and for how long the plant is exposed to the stress (Farooq et al., 2011).

Effects of warming in wheat

Plants can detect temperature by differences in metabolic activity: photosynthesis, membrane fluidity, and protein configuration (Ruelland and Zachowski, 2010; Farooq et al., 2011). Plant response to heat stress involve signaling components such as protein kinases, transcription factors and functional genes such as heat shock proteins and catalase. In addition, hormones such as auxins, salicylic acid and abscisic acid, are also involved in heat response (Qu et al., 2013).

Moderate increases in temperature, in general, can lead to a faster growing rate reducing crop duration (Driedonks et al., 2016). During anthesis, the optimum temperature

for wheat ranges from 12 to 22°C (Farooq et al., 2011). Temperatures above 27°C during anthesis increase the number of sterile grains and potentially affect production (Mitchell et al., 1993, Ferris et al., 1998). Many studies suggest a change in phenology in response to change in temperature. For example, a reduction in pre-anthesis period in wheat was reported when studying increases in air temperature (Hou et al., 2012; Tian et al., 2014). Our group recently published a study using soil cables to heat the rhizosphere of winter wheat plants. We observed that increasing the rhizosphere temperature by ~ 2°C resulted in ~ 3.5 days earlier heading date (Tessmann and Van Sanford, 2018). A study in Germany reported a heading date of winter wheat was 14 days earlier in response to warming (Rezaei et al., 2015). The authors suggested that earlier heading date compensates for the effects of warming temperatures. Prediction models also point for changes in plant phenology. Fels-Klerx et al. (2012), using a climate model predicted an earlier flowering of wheat of about 1 to 2 weeks in Europe. In Australia, Zheng et al. (2012) predicted that warmer winters would shorten the bread wheat growing season by up to 6 weeks leading to a potential reduction in yield. His group suggested utilizing early sowing and longer season varieties as an adaptive strategy.

Despite the predictions pointing to a reduction in yield, a few investigators have found different results. Li et al. (2016) showed that elevated CO_2 and soil warming improved yield in wheat but decreased mineral accumulation such as K, Ca and Mg. In barley, soil warming had no effect on biomass and yield (Högy et al., 2013). Wheat is sensitive not only to increased daytime temperature but to nighttime temperature increases as well. An increase of 1°C in nighttime temperature decreased yield by 10%, and no differences were observed for the same increase in daytime temperature (Lobell et al.,

2005). Prasad et al. (2008) found that temperatures >20°C during the night can reduce spikelet fertility and linearly decrease grain filling duration. A 27% yield decrease in wheat was observed when nighttime temperatures increased by 2.5°C. The authors attributed that result to decreased tiller fertility, reduced number of spikelet and grains per spike (Fang et al., 2010). Another study showed that nighttime temperature had no effect on winter wheat yield (Fang et al., 2012).

Climate change will also affect disease occurrence and distribution. Plant diseases cause around 10-16% in losses globally (Chakraborty and Newton, 2011). One of the effects of climate change is the change in plant phenology by accelerating heading date. In an environment in which the pathogen is present, this change in phenology can enhance disease levels (Boonekamp, 2012). FHB is strongly affected by meteorological factors. A warm and wet environment are required for disease development (Vaughan et al., 2016). Changes in rain intensity and frequency are predicted to happen with climate change (Tataw et al., 2016). Thus, predicting disease occurrence and magnitude will be difficult. For example, in Scotland, FHB is predicted to decrease due to dry conditions during flowering (Skelsey and Newton, 2015). Fels-Klerx et al. (2012), using wheat phenology and DON concentration models, predicted an increase in DON contamination by up to 3 times due to climate change in north-western Europe. Using 238 cultivars from the eastern US in an artificially warmed environment, our group observed an increase in disease levels under warmed conditions (Tessmann and Van Sanford, 2018).

Genome wide association studies

Genome wide association studies (GWAS) have become an important tool to investigate inheritance of traits in large mapping panels. GWAS uses the entire genome. The theory is that the genetic variation that affects a trait is common among unrelated individuals (Visscher et al., 2012). GWAS analysis takes advantage of historical recombination events that happened in diverse populations and lead to quick decline of linkage disequilibrium (Flint-Garcia et al., 2003; Xiao et al., 2017).

Linkage disequilibrium (LD) is nonrandom association between alleles at different loci. In the absence of selection, mutation, or migration, a large population with random mating will be in linkage equilibrium (Falconer and Mackay, 1996; Flint-Garcia et al., 2003; Visscher et al., 2012). LD is important because only markers in strong LD with a trait will be associated with the trait (Flint-Garcia et al., 2003; Myles et al., 2009). Thus, the GWAS strategy is to have enough markers across the genome so the functional allele will likely be in linkage disequilibrium with at least one marker (Myles et al., 2009; Arruda et al., 2016). Phenotypic variation within the population is fundamental for identification of a true association between single nucleotide polymorphism (SNP) and trait (Korte and Farlow, 2013).

The power and resolution of GWAS in self and cross-pollinated species is different. In self-pollinated species, LD decay rate is slower due to self-fertilization and GWAS cannot resolve a single gene, while in cross-pollinated species, a rapid LD decay occurs, which makes GWAS more powerful and able to reach single gene level (Huang and Han, 2014).

Association studies use statistical techniques that measure the strength of the association between a marker and trait (George and Cavanagh, 2015). Many studies have been conducted in recent years in wheat for traits such as: seeding emergence and tiller number (Chen et al., 2017), agronomic and physiological traits in spring wheat (Ogbonnaya et al., 2017), grain yield and related traits (Wang et al., 2017), quality traits in winter wheat (Kristensen et al., 2018), panicle traits (Liu et al., 2018).

Several studies have used GWAS as an approach to identify promising QTL associated with resistance to FHB. Jiang et al. (2015) used 9K and 90K Infinium SNP arrays with 372 European wheat varieties to determine the genetic architecture of FHB. The authors detected the highest number of QTL on high density 90K SNP. Miedaner et al. (2011,) using 455 European soft winter wheat and 115 single sequence repeats (SSR) markers, identified associations on chromosomes 1B, 1D and 2D. Arruda et al. (2016) detected significant SNPs on chromosomes 4A, 6A, 7A, 1D, 4D and 7D for FHB resistance. A GWAS for FHB in an artificially warmed environment detected 10 significant (p<0.001) SNPs in the warmed treatment (Tessmann and Van Sanford, 2018). As pointed out by Jiang et al. (2015), high-throughput GWAS has the potential to answer two quantitative questions i) how many genes are involved in a trait, and ii) the allele distribution at these gene loci.

Identification of QTLs decreases exponentially with increased trait complexity. Thus, when a large number of small effect QTL are involved it is more difficult to find significant QTL. While when a small number of large effect QTL are involved, one is more likely to detect significance (Myles et al., 2009; Massman et al., 2011). A large number of QTL are being identified, however they only account for a small portion of the variation

observed in the phenotype (Xiao et al., 2017), especially in human disease studies (Eichler et al., 2010). Myles et al. (2009) suggested two reason for the 'missing heritability': i) low frequency of functional alleles, where these alleles would have little influence in the whole population and would be difficult to detected in the analysis; and ii) the number of genes involved in a specific trait would affect the power of detection. Brachi et al. (2011) also pointed other reasons for the 'missing heritability': i) rare alleles that are unique and only detected if sampling is adequate; ii) allelic heterogeneity, when multiple functional alleles of the same gene exist and express different phenotypes; iii) variation due to epistatic interaction between genes; iv) epigenetic variation.

GWAS FOR FUSARIUM HEAD BLIGHT RELATED TRAITS IN WINTER WHEAT (*TRITICUM AESTIVUM* L.) IN AN ARTIFICIALLY WARMED TREATMENT

Elisane W. Tessmann and David A. Van Sanford*

Department of Plant and Soil Science, University of Kentucky, Lexington, KY 40546, USA; ewte222@g.uky.edu

*Correspondence: dvs@uky.edu; Tel.:+1-859-338-2409

Received: 13 March 2018; Accepted: 3 May 2018; Published: 5 May 2018 in Agronomy - Basel

doi: 10.3390/agronomy8050068

ABSTRACT

Global temperature increases will affect Fusarium head blight (FHB) levels in wheat (*Triticum aestivum* L.). A pressing question is whether current sources of resistance will be effective in a warmer environment. We evaluated phenotypic response to disease in 238 soft winter wheat breeding lines and cultivars grown in 2015–2016 and 2016–2017 under control and warmed (+3ºC) conditions. Warming was achieved with heating cables buried 3 cm in the rhizosphere. We measured heading date, plant height, yield, FHB rating, Fusarium damaged kernels (FDK), deoxynivalenol (DON), leaf blotch rating, powdery mildew rating and leaf rust rating. There were significant (p<0.01) differences among genotypes for all traits measured. Genome-wide association study (GWAS) identified 19 and 10 significant SNPs in the control and warmed treatments, respectively. FDK and DON levels were often significantly (p<0.05) higher in warmed than in control when we contrasted alleles at important quantitative trait locus (QTL) such as *Fhb1*, *Rht-B1* and *D1* and all vernalization and photoperiod loci. Increased rhizosphere temperature resulted in a significantly (p<0.01) earlier heading date (~3.5 days) both years of the study. Rank correlation between warmed and control treatments was moderate (r = 0.56). Though encouraging, it indicates that selection for performance under warming should be carried out in a warmed environment.

Keywords: artificially warmed treatment; fusarium head blight; deoxynivalenol; fusarium damaged kernels; soft red winter wheat; GWAS; QTL

Introduction

Wheat (*Triticum aestivum* L.) is one of the most important cereals in the nation and widely consumed around the world. Increases in population, changes in diet, social and policy issues especially in developing countries, will increase the demand for staple foods such as wheat (Borlaug and Dowswell, 2013). Among the challenges that researchers are facing, the most daunting is how to increase production in a sustainable way with minimum increase in area. Ray et al. (2013) using historical data determined that wheat yield is increasing by 0.9% per year, substantially lower than the rate that we need (~2.4% per year by 2050). In addition, increased temperature and changes in rainfall patterns over the next decades will require extra effort to increase grain production (IPCC, 2014). Without efficient selection of adapted plants and improvement in genetic material, a global decrease in production is estimated on the order of 6.0% in wheat, 7.4% in maize, 3.2% in rice and 3.1% in soybean for each degree-Celsius increase in temperature (Zhao et al., 2017).

IPCC (2014) projects a global increase, under all emission scenarios, in air temperature by 1 to 3.7ºC by the end of this century. Decrease in crop production due to weather events is not only a future problem, in that drought, flood and extreme temperatures are already reducing production worldwide. Lesk et al. (2016) estimated that from 1964 to 2007 around 9–10% of the reduction in cereal production worldwide was due to drought and extreme heat events. They determined that yield losses due to drought were associated with decreased harvested area and heat events, though extreme heat was the primary factor in yield decreases. In a more regional analysis of data from wheat field trials in the United States from 1985 to 2013, there was a yield reduction due to extreme heat in spring (Tack et al., 2015). In addition, Tack et al. (2015) observed that recently released

cultivars had a lower ability to resist heat stress than old varieties. Climate change is expected to increase not only air temperature but also soil temperature. Studies with winter wheat (Patil et al., 2010) and barley (Högy et al., 2013) testing soil warming conditions showed limited crop development and yield production.

Climate change will also affect disease occurrence, distribution and intensity. A major disease threat to wheat and other small grains is Fusarium head blight (FHB) which causes yield losses, decreases in grain quality and toxin production (Audenaert et al., 2013). Using a modeling approach, Backhouse et al. (2014) found a positive correlation between climate and distribution of pathogenic species of *Fusarium* which includes *Fusarium graminearum*. His group predicted wide distribution in countries where this disease already occurs and further, that new regions such as Mexico, North Africa, Ethiopia and western Siberia would be vulnerable to FHB epidemics (Backhouse, 2014).

Despite the ability of this pathogen to reduce yield, the production of mycotoxins such as deoxynivalenol (DON) which are harmful to plants, animal and humans is the driving force behind selection for resistance to FHB (Audenaert et al., 2013). A study predicting wheat phenology and DON in north-western Europe pointed out that due to climate change, flowering and maturity will be 1 to 2 weeks earlier in the season and DON levels will increase up to 3 times in most of the regions where the study was carried out (Fels-Klerx et al., 2012). Fusarium damaged kernels (FDK) is also an important trait since kernel damage is associated with reduced test weight, which directly affects farmers who receive lower prices for their grain.

Success during infection and colonization of plants by *Fusarium* is a function of host susceptibility, time of infection, fungal pathogenicity and meteorological conditions

(Kugler et al., 2013; Vaughan et al., 2016). The disease occurs during or just after flowering where spores that overwintered in plant debris can germinate and penetrate floral tissue. FHB is driven by weather conditions where wet and warm environments are required for fungal development (Audenaert et al., 2013). Integrated management using cultivar resistance and FHB-specific fungicide application are the most effective management techniques (Cowger et al., 2016). The challenges of fungicide application derive mainly from timeliness of the fungicide application. Prediction tools such as FHB Prediction Center (http://www.wheatscab.psu.edu) are helping farmers to assess the risk of disease and decide whether or not fungicide application is profitable.

In recent decades, investigators worldwide have carried out extensive studies to develop genetically resistant cultivars. Quantitative trait locus (QTL) such as *Fhb1*, *Qfhs.ifa-5A* and *QFhs.nau-2DL* are widely used as sources of resistance in breeding programs (Kugler et al., 2013; Zhuang et al., 2013; Clark et al., 2016). *Fhb1*, for instance, provides resistance against spread of the disease (type II resistance) while *Qfhs.ifa-5A* provides resistance to penetration (type I resistance) (Buerstmayr et al., 2002, 2003). A map-based cloning of *Fhb1* was recently published, opening the possibility of direct cloning and use of that gene (Rawat et al., 2016). Furthermore, techniques such as RNA interference (RNAi) for fungicide development and cultivar resistance are promising approaches to controlling FHB (Machado et al., 2017). However, FHB is a quantitative disease, thus an individual QTL approach may not be efficient in controlling this disease, since resistant cultivars have a compilation of major and minor genes that work together to provide resistance.

Plant resistance to diseases will be positively or negatively affected by climate change (Skelsey and Newton, 2015; Fels-Klerx et al., 2016). The genetic composition of a variety can provide resistance to disease; however, environmental conditions largely influence whether or not resistance genes will be expressed (Vaughan et al., 2016). This raises an important question: how responsive are our cultivars to climate change? Are the current sources of resistance to FHB responsive to an increase in temperature? To our knowledge, there are no studies evaluating whether the current sources of FHB resistance will be effective in a warmed environment. In order to answer that question, we conducted over two years an artificially warmed experiment in order to assess the disease response in a large, diverse wheat mapping panel. Our goal was to: determine whether or not the QTL used today for FHB resistance would be responsive to disease in an artificially warmed treatment. Specific objectives were: (i) to evaluate phenotypic response to FHB and other disease traits in a large, diverse soft wheat mapping panel under warmed and control conditions; and (ii) to determine whether there were QTL associated with FHB traits under warmed conditions when compared with control conditions based on GWAS analysis.

Materials and Methods

Site description and experimental design

The study was conducted at the University of Kentucky Spindletop Research Farm in Lexington, KY (38◦7'37.81" N, 84◦29'44.85" W). Soil type at the site is a Maury silt loam (fine, mixed, semi active, mesic Typic Paleudalfs). The experimental material consisted of two hundred and thirty-eight elite soft red winter wheat cultivars and breeding

lines from an elite mapping panel constituted under the Triticeae Coordinated Agricultural Project (TCAP). The TCAP project was a consortium that involved 21 states and 55 universities, funded by the National Institute for Food and Agriculture (NIFA) of the United States Department of Agriculture (USDA; http://www.triticeaecap.org/). Populations such as the elite panel were genotyped with the 90 K Illumina SNP chip.

The two hundred and thirty-eight wheat lines used in this study differed in characteristics such as heading date, height and the environment to which they were adapted. The studies were planted 20 October 2015 and 22 October 2016 in a nested factorial split block design at Spindletop farm in Lexington, KY. The experimental unit was a hill plot. Two replications per genotype per treatment were planted in 2015 whereas four replications were planted in 2016. Two treatments were used: the control (ambient) treatment and an artificially warmed treatment. Soil heating cables (Gro-Quick 42 m length, 120 V, 700 watt) were used to warm the rhizosphere in the artificially warmed treatment (Hitz, 2015; Russell, 2017). Cables were active during the majority of the growing season, from December through June.

Cables were buried at a depth of ±3 cm between rows to warm the rhizosphere. A Campbell weather station was placed at the site to measure soil temperature and air temperature within each treatment. There were 8 thermocouple wires (OMEGA Engineering, Stamford, CT, USA) in each block in the warmed treatment and two in the control treatment that measured soil temperature. Soil temperature sensors were placed at a depth of ±5 cm. Air temperature was measured by two thermocouple wires in the warmed treatment and two in the controlled treatment. The air sensors were positioned on a metal bar placed into the ground and they were moved up to follow plant growth. Each probe

within each treatment measured soil/air temperature daily every 15 min and measurements were averaged along each row throughout the duration of the study. Each row was flanked by two heating cables. The two temperature sensors for each row were averaged to determine the temperature threshold for each cable compared to the control treatment. When the temperature difference between control and warmed was less than 5ºC, cables in the warmed treatment were activated to heat the soil.

In 2016, hill plots were sprayed with a conidial spore solution to encourage initial infection of wheat plants by *F. graminearum*. Spore concentration in the solution was 50,000 macroconidia mL^{-1}. Hill plots were sprayed manually with a bottle sprayer before, during and after flowering to insure the presence of spores during the most favorable stage for infection. In 2017, scabby corn (*Zea mays* L.) seed infected with *F. graminearum* spores was spread three weeks prior to heading of the earliest genotype (Balut et al., 2013). Inoculum came from 27 isolates taken from scabby wheat seed collected from multiple locations across Kentucky, 2007–2010. An overhead irrigation system on an automatic timer was used to provide favorable conditions for disease development.

Traits measured

Heading date (HD; Julian) was determined for each hill plot when more than 50% of the spikes had emerged from the flag leaf. Plant height (PH; cm) was measured from the soil surface to the top of the spike, excluding awns. FHB rating is a visual index of FHB incidence and severity ranging from 1 (≤10% of spikes showed FHB symptoms) to 9 (≥90% of spikes showed FHB symptoms) 24 days after heading date. Yield from each hill plot was recorded in grams m^{-2}.

In addition to the traits described above, in 2017 FHB pressure was sufficiently high that we were able to measure Fusarium damaged kernels (FDK) and deoxynivalenol (DON). In addition, there was natural infection by other pathogens such that we were able to rate leaf blotch (*Septoria tritici*), powdery mildew (*Blumeria graminis* f. sp. *Tritici*) and leaf rust (*Puccinia triticina*). FDK is a visual estimate of kernel damage ranging from ≤5% to ≥90% of scabby kernels. Two samples of each genotype were sent for DON analysis; sample 1 was composed of replications 1 and 2 and sample 2 was composed of replications 3 and 4. Each whole kernel sample (15 g) was sent to University of Minnesota DON testing laboratory which uses gas chromatography with mass spectrometry (GC-MS) (Mirocha et al., 1998). For leaf blotch, powdery mildew and leaf rust rating a visual estimate was taken ranging from 1 (≤10% of plants showed disease symptoms) to 9 (≥90% of plants showed disease symptoms). At harvest maturity, all spikes of each hill plot were hand harvested using a sickle. Harvest maturity was determined when grain was hard and could not be split by a thumbnail. After harvest, grain was mechanically threshed.

Statistical analysis

Analysis of variance (ANOVA) was performed using SAS Procedure GLM (SAS Institute Inc., 2011) to determine genotype and treatment effects. The model used was:

$$Y_{ijkl} = \mu + T_i + R(T)_{ij} + G_k + T_i x G_k + Y_l + T_i x Y_l + G_k x Y_l + \varepsilon_{ijkl}$$

where Y_{ijkl} = the observation in the kth genotype in the *jth* rep in the *ith* treatment, μ = the overall mean, T_i = the effect of the *ith* treatment, $R(T)_{ij}$ = the effect of *jth* rep within *ith* treatment, G_k = the effect of the kth genotype, $T_i \times G_k$ = the effect of the interaction of the *ith* treatment and the kth genotype, Y_l = the effect of the lth year, $T_i \times Y_l$ = the effect of the

interaction of the *ith* treatment and the lth year, $G_k \times Y_l$ = the effect of the interaction of the kth genotype and the lth year, ε_{ijkl} = the residual error.

Broad sense heritability of the traits measured in both years of the study was estimated on an entry mean basis using the following model:

$$Y_{ijk} = \mu + Y_i + R(Y)_{ij} + G_k + Y_i x G_k + \varepsilon_{ijk}$$

where Y_{ijk} = the observation in the kth genotype in the *jth* rep in the *ith* treatment, μ = the overall mean, Y_i = the effect of the *ith* year, *R(Y)ij* = the effect of *jth* rep within *ith* year, G_k = the effect of the kth genotype, $Y_i \times G_k$ = the effect of the interaction of the *ith* year and the *kth* genotype, ε_{ijk} = the residual error. Because we wanted to determine whether heritability was affected by warming, estimates were generated for each treatment separately.

Data was analyzed using the GLM procedure of SAS (SAS Institute Inc., 2011). Genotypic and phenotypic variance components were estimated from the expected mean squares (EMS) and heritability estimates were computed as:

$$h^2 = \frac{\sigma_g^2}{\sigma_p^2}$$

where h^2 = heritability, σ_g^2 = genotypic variance, σ_p^2 = phenotypic variance. Confidence intervals (90%) were calculated after Knapp et al. (1985) as:

$$UL = 1 - \left(\frac{MS3}{MS2} x\ FUL\ (0.05, v1\ and\ v2\ df)\right)^{-1}$$

$$LL = 1 - \left(\frac{MS3}{MS2} x\ FLL\ (0.95, v1\ and\ v2\ df)\right)^{-1}$$

where UL = upper limit of the confidence interval, MS3 = entry mean square, MS2 = residual mean square, FUL and FLL = F value for the upper and lower limits, respectively.

Several traits were measured only in 2016–2017; broad sense heritability estimates of these traits are termed "repeatability" (Holland and Nvquist, 2003) and the model used for estimation was:

$$Y_{ij} = \mu + R_i + G_j + \varepsilon_{ij}$$

where effects are defined as above.

Proc CORR (SAS Institute Inc., 2011) was used to analyze the relationships among traits on an entry mean basis

Genome Wide Association

All entries in the mapping panel were genotyped with the 90 K Illumina SNP chip to identify single nucleotide polymorphisms (SNPs). A total of 3919 SNPs were used for the GWAS in this study. Genotypic and phenotypic data were formatted to HapMap and text file format, respectively. We used the following QTL as covariates in the model: *Fhb1*, *Rht-B1*, *Rht-D1*, *Vrn-A1*, *Vrn-B1*, *Vrn-D3*, *Ppd-A1*, *Ppd-B1* and *Ppd-D1*. Genomic Association and Prediction Integrated Tool (GAPIT; Lipka et al., 2012) which uses compressed mixed linear model approach for the genome wide association study was used to identify SNPs associated with the traits of interest.

Results

Phenotypic variation

The average soil temperature over the whole growing season was higher in the warmed treatment than in the control treatment for both years of this study. The cables

were programmed for a 5ºC temperature threshold, however the temperature in the warmed treatment ranged from 0.65–2.95ºC and 0.75–3.30ºC greater than the control treatment in 2016 and 2017, respectively (Figure S2.1a, b).

Means for control and warmed treatment in 2016 and 2017 are presented in Table 2.1. Independent of the warming treatment, heading date was 8.4 days earlier in 2017 than in 2016, on average. The warmed treatment was 3.53 and 3.41 days earlier, on average, when compared with the control treatment in 2016 and 2017, respectively. Intensity of FHB differed dramatically between the years of the study. In 2016 the experiment was sprayed with conidia 3 times; however, the lack of moisture reduced scab pressure to the extent that we did not obtain reliable data for FDK and DON. In 2017, we were able to use scabby corn and mist irrigation and had a much higher level of disease pressure. Thus, of the scab traits, only FHB rating was recorded both years of the study; DON and FDK data are presented only for 2017. For FHB rating the difference between control and warmed treatment in 2016 was 0.24, while in 2017 was 0.55. Plant height in the warmed treatment was slightly greater (0.73 cm), on average, than in the control in 2016. In contrast, the warmed treatment reduced plant height 5.74 cm in 2017 (Table 2.1). Both increased and reduced height in response to warming have been observed in other studies carried out in our lab (Hitz, 2015; Russell 2017). Yield was evaluated in small hill plots thus its estimation is not as reliable as estimates based on conventional yield plots (Hall and Van Sanford, 2003). Yield increased under warmed conditions by 21% in 2016 and decreased by 6.4% under warmed conditions in 2017. For the disease traits such as DON, FDK, leaf blotch rating and powdery mildew rating the warmed treatment increased disease pressure. DON increased by 84%, FDK 131%, leaf blotch rating 17.3% and powdery mildew rating

0.66% under warmed conditions. For leaf rust rating, there was no change between control and warmed treatments (Table 2.1).

The warming treatment had a significant (p<0.05) effect on HD, DON, FDK and leaf blotch rating (Table 2.1). There were significant differences among the genotypes for all traits evaluated. Significant treatment × genotype interaction (p<0.05) was observed for DON, FDK, leaf blotch rating and leaf rust rating (Table 2.1). The results of the interactions involving year are shown only for the traits evaluated in both years: HD, FHB rating, PH and yield. While there was no significant year × genotype interactions, year × treatment interaction was significant for all traits, except for HD (Table 2.1).

Correlations among traits under control and warmed conditions for 2016 and 2017 are shown in Tables 2.2 and 2.3, respectively. Low correlations among most traits were observed in 2016 (Table 2.2). Heading date was positively correlated with plant height in both treatments. FHB rating was negatively correlated with plant height: −0.28 under warmed conditions and −0.20 under control conditions (p<0.01). A negative correlation was also observed between FHB rating and yield in both treatments. Yield and plant height were positively correlated in both years under control and warmed conditions (Tables 2.2 and 2.3); correlations were intermediate in all cases.

In 2017, the correlation between HD and FHB rating varied among treatments; in the warmed treatment, the traits were positively correlated (0.51), while under control conditions there was no correlation between these traits (Table 2.3). Under control conditions, PH and FHB rating had a correlation of −0.30, however under warmed conditions the correlation was not significant. FDK and DON were correlated with values of 0.42 and 0.57 under control and warmed conditions, respectively. Leaf diseases were

not correlated with FDK and DON, with the exception of a weak but significant correlation between powdery mildew rating and FDK in the warmed treatment (−0.12; Table 2.3).

Heritability estimates and confidence intervals (90%) are presented in Table 2.4. These estimates were based on individual analysis of treatments. For traits measured in both years heritability ranged from 0.23 to 0.81 in the control treatment and from 0.13 to 0.80 in the warmed treatment (Table 2.4). Repeatability of those disease traits measured only in 2017 ranged from 0.39 to 0.80 in the control treatment and 0.35 to 0.84 in the warmed treatment (Table 2.5). Repeatability of FDK was 0.80 and 0.84 under control and warmed conditions, respectively. DON also had high repeatability in both treatments, with values of 0.71 for control and 0.75 for warmed treatments. Leaf blotch rating had repeatability of 0.39 and 0.35 under control and warmed conditions, respectively. A repeatability estimate of 0.42 was observed for powdery mildew rating under control conditions versus 0.69 under warmed conditions (Table 2.5). Heritability of rating had a confidence interval that enclosed zero in the warmed treatment (Table 2.4). For traits such as heading date and plant height high heritability values were observed in control and warmed treatments (Table 2.4).

Genome Wide Association Study (GWAS)

We performed a GWAS for the control and warmed treatments separately and used known QTL as covariates in the model. Manhattan plots for FDK, DON, HD and PH show promising QTL for those traits (Figure 2.1). Potential SNPs for FDK, DON, HD and PH are presented in Table 6. Only SNPs with LOD score >3 are listed in the table. We detected 19 SNPs in the control and 10 SNPs in the warmed treatments. These SNPs were located

on almost all chromosomes with the exception of 2B, 2D, 4B, 4D, 6D and 7D where we did not detect SNPs associated with the traits measured in this study (Table 2.6; Figure 2.1). Seven SNPs in the control treatment were found for FDK on chromosomes 1A, 1B, 2A, 3B and 5A. The SNP effects ranged from −2.32 to 2.40%. Two SNPs on chromosome 1B, M1905 and M1563, explained 2.40 and 2.11%, respectively, of the variation observed for FDK under control conditions (Table 2.6). The SNPs M1591 and M480 were associated with 1.99 and 1.72%, respectively of variation in FDK. There was no SNP under warmed conditions for FDK that met our LOD score threshold.

The association mapping for DON identified five SNPs in the control treatment and three SNPs in the warmed treatment. SNPs, under control conditions, were observed on chromosomes 1B, 4A, 6A and 6B, while under warmed conditions chromosomes 3B and 4A had SNPs for DON. A SNP (M1528) at position 243.59 on chromosome 1B had a positive effect of 0.87% on the variation observed for DON under control conditions. A negative effect of −0.96% was associated with SNP M11423 on chromosome 6B under control conditions (Table 2.6). Under warmed conditions a SNP (M5744) on chromosome 3B explained 1.13% of the DON variation (Table 2.6).

Heading date had four and six SNPs for control and warmed treatments, respectively. Under control conditions, SNPs were observed on chromosomes 4A, 5D and 7B with effects ranging from -0.55 to 0.62%. Under warmed conditions, chromosomes 1D, 3B, 3D and 7A had SNPs where the effects ranged from −0.78 to 0.68% for HD (Table 2.6). The GWAS for plant height uncovered four potential SNPs on chromosome 5B, three in the control and one in the warmed treatment (Table 2.6). Under control conditions SNPs

M8584 and M9257 had effects of 2.41 and 2.13%, respectively. The SNP M8584 was also identified in the analysis for the warmed treatment and it explained 2% of the variation.

The effects of known QTL in the genotype response to an artificially warmed treatment for FDK and DON are presented in Table 2.7. The QTL analyzed in the population were: *Fhb1*, *Rht-B1*, *Rht-D1*, *Vrn-A1*, *Vrn-B1*, *Vrn-D3*, *Ppd-A1, Ppd-B1* and *Ppd-D1*. The population was classified for each allelic form of the QTL and the levels of FDK and DON are presented for each treatment. In the warmed treatment, there was drastically increased disease pressure. For example, FDK increased from 98 (*Vrn-A1-short*) to 135% (*Ppd-A1-sensitive*) in the warmed treatment when compared with the control treatment. DON levels increased under warming conditions from 50 (*Rht-B1b*) to 121% (*Vrn-A1-short*) when compared with the control treatment (Table 2.7). Under warming, lines with resistance alleles at *Fhb1* had 13.08 and 29.87% FDK under control and warmed conditions, respectively, an increase of 128% associated with warming. For DON, on the other hand, these lines did not differ significantly from one treatment to the other. Lines that did not possess *Fhb1*-R alleles showed 120% more FDK and 69% more DON under warmed conditions (Table 2.7).

In our population, the dwarfing alleles, *Rht-B1b* and *Rht-D1b*, were present in 123 and 84 genotypes, respectively. Wild type alleles, *Rht-B1a* and *Rht-D1a*, were present in a total of 112 and 151 genotypes had those genes, respectively. Both alleles at the *Rht* loci were strongly affected by the treatment, in that the warmed treatment changed plant morphology by shortening the plants (Figure 2.2). FDK levels were 129 and 112% greater for *Rht-B1b* and *Rht-D1b* under warmed conditions, respectively, than under control conditions (Table 2.7). Under warming, DON levels for *Rht-B1b* and *Rht-D1b* were

increased 50 and 78%, respectively, over control levels. Average levels for FDK and DON were also higher under warmed than under control conditions for the wild-type genotype (*Rht-B1a* and *Rht-D1a*).

The TCAP panel was also genotyped at vernalization loci. A total of 225 genotypes had *Vrn-A1*, 230 had *Vrn-B1* and 165 genotypes had *Vrn-D3* (Table 2.7). In the warmed treatment, genotypes with *Vrn-A1* had 123% more FDK and 60% more DON than in the control treatment. Similarly, *Vrn-B1* had increase of 122% for FDK and 62% for DON under warmed conditions. The last *Vrn* gene, *Vrn-D3*, was also associated with high DON concentration and high FDK level under warming, with increases of 120% for FDK and 66% for DON in the warmed treatment versus the control. In addition to the vernalization genes, we also analyzed disease impact as affected by photoperiod genes *Ppd-A1*, *Ppd-B1* and *Ppd-D1*. A total of 138 genotypes were *Ppd-A1-insensitive*; these showed an increase in FDK of 111 and 60% for DON under warmed conditions (Table 2.7). In the nineteen genotypes with the *Ppd-B1-insensitive* allele, FDK increased by 109% and DON concentration was 65% higher in the warmed treatment when compared with the control. Genotypes with *Ppd-D1-insensitive* alleles had increased disease levels of 119 and 65% for FDK and DON, respectively, under warmed conditions. Disease impact for *Ppd* sensitive genotypes was also higher in the warmed treatment when compared to the control: FDK increased by 135, 121 and 125% for *Ppd-A1*, *Ppd-B1* and *Ppd-D1*, respectively. DON levels were 64, 61 and 62% higher in the warmed treatment than in the control for *Ppd-A1*, *Ppd-B1* and *Ppd-D1*, respectively (Table 2.7).

Discussion

Maintaining and/or increasing yield production under climate change is one of the most important challenges of this century. The stress caused by elevation in temperature, changes in rainfall patterns and increases in pests and diseases is predicted to affect crop production significantly (Lesk et al., 2016; Vaughan et al., 2016; Zhao et al., 2017). Field experiments can best assess crop response to the environment due to natural exposure to pest and disease pressure and weather conditions such as rain, temperature and cloud cover. In this study, we simulated increased temperature by artificially warming the rhizosphere and by causing an FHB epidemic, with the goal of assessing changes in plant response under these stresses. We observed that increasing the temperature by 1.8 and 2.0ºC in 2016 and 2017 respectively, reduced the length of the winter wheat growing period (Table 2.1). Other researchers have reported field studies simulating increases in air temperature that showed an effect on wheat phenology by reducing the pre-anthesis period (Hou et al., 2012; Tian et al., 2014). In another study, soil warming conditions have shown a shortening of the total crop growing season in wheat (Patil et al., 2010).

Understanding the effects of a warmed environment in crop response is fundamental to achieving sustainable production in the years ahead. Crop models have been used to predict the effects of climate change. These models show a decrease in yield for each degree-Celsius increase in temperature (Fels-Klerx et al., 2012; Liu et al., 2016; Zhao et al., 2017). Our results showed an increase in yield of 21% in 2016 and a decrease of 6.4% in 2017 under warmed conditions. Increased yield in 2016 could be explained by the experimental design where in 2016 we had only two replications while in 2017 we had four. Since we presented average values for the genotypes in hill plots, the number of

replications could affect the final value. Even though crop models predict a reduction in yield in a warmer environment, other researchers have also found an increase in yield. Li et al. (2016), evaluating the effects of soil warming in wheat, observed an increase in yield. Similar results were observed by Tian et al. (2014) in an experiment with air temperature increase tested in winter wheat. Högy et al. (2013) under elevated soil temperature observed no change in grain yield of barley. Variation in yield response to warming agrees with previous studies in our group (Russel, 2017).

Progress in plant breeding depends, in part, on the heritability of the traits of interest. We were particularly interested in differences in heritability estimates between control and warmed conditions. As expected, based on previous studies (Russell, 2017), heading date and plant height had high heritability in the control treatment as well as in the warmed treatment. FHB rating, an indication of disease incidence and severity, had a low heritability in the control environment and its heritability under warmed conditions was not significantly different from zero. Heritability of rating in one of our advanced breeding line trials (grown in single row plots in the scab nursery) in 2016 and 2017 was low, averaging 0.32 (data not shown). The other factors that can account for the low heritability of FHB rating in the present study are: (i) hill plots present a different picture to the person rating than do single rows—there is not as much material for the reviewer to look at and rate; and (ii) the very low h^2 rating in the warmed treatment is probably a reflection of the more rapid development of the plant, making it even more difficult to rate at the proper time.

For FDK and DON, repeatability estimates were greater in the warmed treatment than in the control treatment (Table 2.5). FDK repeatability estimates were 0.80 and 0.84

under control and warmed conditions, respectively. Similarly, DON repeatability estimates were 0.71 and 0.75 under control and warmed conditions, respectively. High repeatability values indicated reproducibility of the data in the control and warmed treatment. Our results suggest that selection for disease traits such as FDK and DON could be achieved in a warm treatment. Heading date and FHB rating had a moderate correlation of 0.51 in the warming treatment (Table 2.3). This result implies that genotypes with a long flowering period had a prolonged period of exposure to the pathogen.

Genome wide association studies (GWAS) provide a tool that breeders can use to investigate a large population of breeding lines and cultivars for association of genomic markers with important agronomic and disease traits. Furthermore, GWAS, by concurrent analysis of genotypic and phenotypic data, allows the detection of QTL across populations with different backgrounds (Lee et al., 2017; Ogbonnaya et al., 2017). Exploring recombination events that occurred years ago in unrelated individuals to identify alleles in linkage disequilibrium with the marker are one of the advantages of GWAS (George and Cavanagh, 2015; Arruda et al., 2016). The panel used in this study represents soft red winter wheat lines and cultivars from breeding programs distributed across 14 states. A large and diverse panel provides a more realistic assessment of the genetic response to a warmed treatment than would be possible with a single bi-parental population. Several breeding lines and cultivars from the panel have been used as parents in the University of Kentucky breeding program; thus, the results of the GWAS are relevant to possible breeding progress for performance in a warmer treatment in our program.

GWAS was carried out for the control and warmed treatments separately. Using a LOD threshold of 3, we detected 19 and 10 SNPs under control and warmed conditions,

respectively (Table 2.6, Figure 2.1). There were significant SNPs for FDK under control conditions with effects ranging from −2.32 to 2.40 on chromosomes 1A, 1B, 2A, 3B, 5A (Table 2.6). However, there was no significant SNP for FDK under warmed conditions. In the control treatment, we observed a significant SNP on chromosome 1B with effect of 0.87% on DON levels was founded. In the warmed treatment, the chromosome 3B had two SNPs with effects of 1.13% and 0.94% on DON; neither of these SNPs is associated with Fhb1. The analysis of HD showed effects ranging from −0.78 to 0.68% under warmed conditions. GWAS revealed potential plant height QTL (not associated with the *Rht* loci) with effects from 1.75 to 2.41%. SNP M8584 was detected in the control and warmed treatment, suggesting that this SNP could be used to evaluate populations under warmed conditions.

In complex diseases such as FHB, major and minor genes are involved in conferring levels of resistance (Buerstmayr et al., 2009; Cai et al., 2016). Plant morphology, resistance to infection and spread of disease as well as environmental factors are critical in determining plant response to pathogens (Buerstmayr et al., 2002, 2003; Srinivasachary et al., 2009). The detection of large QTL effects decreases exponentially with the increase in trait complexity (Robertson, 1967). Thus, for complex traits, with multiple genes involved, an identification of small QTL effects is more likely (Robertson, 1967; Massman et al., 2011). As pointed out by Massman et al. (2011), major QTL in breeding germplasms are under strong selection for multiple years and, thus, fixed in multiple genetic backgrounds. In our study, QTL effects were small, explaining ~−2.5% to +2.6% of the phenotypic variation observed for the traits. If these estimates are accurate, these small effect QTL could be useful in a genomic selection program under warmed conditions.

In addition to the GWAS analysis, we were interested in evaluating the effects of a warmed treatment for disease levels of important wheat QTL (Table 2.7). *Fhb1* is widely used in breeding programs to provide resistance to FHB (Rawat et al., 2016; Steiner et al., 2017). In our population of study, under control conditions the presence or absence of this QTL was not statistically significant, which indicates that in these lines the *Fhb1* resistance was not expressed. *Fhb1* increased by 120 and 128%, FDK levels in warmed conditions in absence and presence of the R alleles, respectively. In addition, lines which lacked the resistance alleles at *Fhb1* presented ~69% increase in DON levels under warmed conditions. Traits such as FDK and DON are very important in selecting for FHB resistance since they quantify visually and chemically the level of infection. These traits express phenotypically the effects of resistant QTL such as *Fhb1*. Heritability estimates are important in guiding the breeder during selection. The high repeatability values across both environments for FDK and DON are encouraging for selection in a warmed environment (Table 2.5).

The semi-dwarfing genes (*Rht-B1* and *Rht-D1*) had an effect on FDK and DON under warmed conditions. A total of 123 lines had *Rht-B1b* (the height reducing allele) in their background. Disease levels for those lines, increased by ~129% and 50% for FDK and DON, respectively, under warmed conditions. Genotypes with *Rht-D1b* also showed increased disease levels of ~112% for FDK and ~78% for DON in the warmed treatment (Table 2.7). Similar results were observed for tall plants with the wild-type alleles (*Rht-B1a* and *Rht-D1a*) where the disease increased under warmed conditions (Table 2.7). The literature suggests that taller genotypes could maintain a cooler canopy in environments with increased temperatures (Pask et al., 2014). A correlation between increased plant

height and reduced FHB is reported in numerous studies (Mao et al., 2010; Buerstmayr et al., 2011; Lu et al., 2013). In one study, the investigators showed that plants with *Rht-B1b* and *Rht-D1b* significantly decreased resistance to initial infection (Type 1) and *Rht-B1b* increased resistance to spread of the fungus (Srinivasachary et al., 2009). Under control conditions, in our study, we observed increased disease when the height reducing alleles were present at these QTL. However, the warmed treatment more than doubled the disease rates independent of the allele present (Table 2.7). A warmer rhizosphere changed plant morphology by shortening the genotypes which could have favored increased disease levels (Figure 2.2). Short plants have higher disease levels due to the microclimate around the spike where high moisture and humidity and close proximity to the inoculum are favorable for increased disease (Klahr et al., 2007). Yan et al. (2011) demonstrated this result by studying near-isogenic lines for *Rht* genes. His group observed that tall plants were more resistant to infection (type I) than the semi-dwarf phenotypes, however when both phenotypes were at the same height that difference disappeared (Yan et al., 2011).

Wheat is cultivated in a wide range of environments due to its vernalization and photoperiod genes. A prerequisite for winter wheat is the accumulation of cold temperature which may be affected by climate change in that warm winters will be more frequent. Vernalization requirements are controlled by *Vrn* genes and specific environmental conditions are needed for the activation of these genes (Allard et al., 2012; Wu et al., 2017). Zheng et al. (2012) studied the rates of climate change in Australia; they suggested an earlier sowing to escape frost and heat stresses. In addition to earlier sowing, longer season varieties have been proposed as a strategy to adapt to future climate change (Zheng et al., 2012, 2016). However, longer season varieties in an environment with a high level of

disease pressure could increase FHB rates due to increased exposure to the pathogen. Meteorological factors such as wet and warm environments are essential for pathogen development and whether or not such factors will occur in a specific region is difficult to predict (Vaughan et al., 2016). In this study, we found that plants with *Vrn-A1* had 123% more FDK and 60% more DON under warmed conditions (Table 2.7). Similar results were observed for *Vrn-B1* with vales of 122% for FDK and 62% for DON. In addition, FDK and DON had increases of 120% and 66%, respectively, for *Vrn-D3*. An analysis of heading date showed that plants under warmed conditions headed earlier than the control (Table 2.1). This suggests that plants were exposed earlier to the pathogen increasing the period for disease, which could explain the increased disease levels found in this study.

Photoperiod (*Ppd*) genes are another important major gene for the transition from vegetative stage to reproductive stage in plants (Guedira et al., 2016). In photoperiod response, the dominant allele confers flowering under short day-length through photoperiod insensitivity (Griffths et al., 2009). After vernalization requirements are met, plants with photoperiod insensitivity will shift to the reproductive stage when temperatures increase, while for photoperiod sensitive plants, a long day is also needed (Guedira et al., 2016). In the warmed treatment genotypes had an earlier flowering period when compared with the control (Table 2.1). An increase in disease levels in the warmed treatment was observed, ranging from 109 to 135% for FDK and from 60 to 65% for DON in the genotypes classified in sensitive and insensitive photoperiod (Table 2.7). An earlier heading period favored disease development since the pathogen was present and wet conditions prevailed in the environment. Therefore, a late heading type, characterized by *Ppd* sensitivity, probably would not avoid disease in a warmer environment.

The ultimate goal in breeding programs is to develop cultivars with high yield performance and adaptability to multiple environments. Screening for agronomic as well as disease resistance traits is fundamental for the selection of superior genotypes. Future projections of increasing temperature add another degree of complexity to selection. Disease evaluations are complex and highly dependent on environmental conditions. Changes in temperature and rainfall patterns can potentially influence levels of disease, with increased spore production and aggressiveness of the pathogen (Vaughan et al., 2016). The design of experiments evaluating increases in temperature associated with disease pressure is critical for a better understanding of the genetics behind the phenotypic response. Moreover, the evaluation under warmed conditions of mycotoxin production such as DON is essential for food security.

The Food and Drug Administration (FDA, 2010) advisory level for DON in finished wheat products such as flour, bran and germ consumed by humans is 1 ppm. Therefore, it is extremely important to develop genotypes that have the ability to fight the infection caused by the pathogen either by morphological characteristics or by resistance QTL (Buerstmayr et al., 2009; Lu et al., 2013). To assess the variation among cultivars and breeding lines for DON levels, we classified the genotypes in the warmed treatment for best and worst performance for DON levels and ranked them from one to two hundred and thirty-eight. After that, we determined how those genotypes performed in the control treatment and their rank order among the 238 genotypes (Table 2.8). Spearman's rank correlation between warmed and control was 0.56, indicating a moderate correlation between genotype ranks in the two treatments.

The best fifteen performers under warmed conditions had, on average, 2.30 ppm DON with the lowest being 1.70 ppm and the highest 2.78 ppm (Table 2.8). Under control conditions, those genotypes had a similar response with 2.48 ppm of DON, on average. The best fifteen in the warmed treatment, had similar performance in the control treatment, with low DON levels. The exception was the genotype 0570A1-2-39-5, which had 135% more DON under control conditions than in the warmed treatment. The best four lines in the TCAP population had levels of DON below 2 ppm in the warmed treatment. Even though the best fifteen lines had DON levels above the FDA advisory level, those lines performed well under warmed conditions and could be indicated for use in breeding programs targeting disease resistance under climate change.

The worst fifteen performing genotypes for DON under warmed conditions and their respective performance under control conditions are presented in Table 2.8. The average DON level was 16.74 ppm under warmed conditions and 8.23 ppm under control conditions. DON levels varied from 14.18 to 24.83 ppm under warming and from 3.75 to 14.55 ppm in the control treatment. These results show the dramatic effect of a warmed treatment on genotypes that have low plasticity to an increase in temperature. Genotypes such as MD03W61-11-2 and BECKER had low levels of DON under control conditions; however, under warmed conditions these two lines showed increases of ~290 and 417%, respectively (Table 2.8). Similarly, the breeding line VA10W-140 had an increase of ~231% under warmed conditions when compared with its performance under control conditions. Some genotypes had high levels of DON in both treatments. For instance, the MO100535 and 03633A1-69-2-5 had 11.90 and 14.55 ppm under control conditions, respectively. Under warmed conditions, MO100535 had 14.58 ppm and 03633A1-69-2-5

had 19.27 ppm, an increase of ~23 and 32%, respectively. For OH08-149-11 and MD03W665-10-3, DON levels were 10.50 ppm and 10.85 ppm under control conditions, respectively. Under warmed conditions, OH08-149-11 and MD03W665-10-3, had 14.63 ppm and 15.23 ppm an increase of ~39 and 40%, respectively. These genotypes presented high levels of disease and thus would not be indicated for use in breeding programs irrespective of treatment.

As mentioned by Atlin et al. (2017), breeding programs will play a fundamental role in adaptation to climate change, where favorable alleles to environmental stresses will need to be rapidly and constantly incorporated into breeding material to produce genotypes capable of adapting to the environment. Therefore, evaluating our current germplasm for disease resistance under warmed conditions is very important. An artificially warmed treatment can be efficient in screening the genotypes and providing valuable information regarding disease response. In the current study, we demonstrated that an increase in soil temperature of a few degrees resulted in earlier heading. As a consequence, disease levels were higher in the warmed treatment than in the control treatment. Our GWAS analysis identified 19 SNPs in the control treatment and 10 SNPs in the warmed treatment. These SNPs can be useful for selection under warmed conditions. We studied the effect of genotype at important QTL such as *Fhb1*, *Rht*, *Vrn* and *Ppd* on disease levels in the population. While we did observe some differences between mutant and wild type alleles at certain loci (e.g., *Rht-D1*), the much greater difference in disease levels was observed between the warmed and control treatments, independent of the allelic form present. Evaluation of cultivar/breeding line performance under warmed conditions showed that the superior genotypes under control conditions were often the best performers in the warmed

treatment. There were exceptions to this trend, however. For example, KY02C-2215-02, 0570A1-2-39-5, 0513A1-1-3 ranked highly for DON under warming but slipped to ranks 51, 186 and 57 in the control treatment (Table 2.8). Falconer (1960) showed that for indirect selection to be superior to direct selection, the genetic correlation must be high and heritability of the trait to be selected must exceed that of the other trait. Using phenotypic correlation as a proxy for genetic correlation and taking the square root of the reliability estimates allows us to estimate Q, the ratio of indirect to direct selection which is expressed as the product of the genetic correlation coefficient and the ratio of the square roots of the heritabilities (e.g., $Q = rg \times h1/h2$; Falcone, 1960). In this instance $rp = 0.58$, $h1 = 0.84$ and $h2 = 0.86$ which leads to a product of 0.56, which is less than 1 and clearly does not support indirect selection under control conditions for performance under warming.

These findings coupled with moderate to high reliability estimates suggest that breeding for disease resistance in a warming treatment should be possible. The active warming method described herein should provide breeders with a tool to pursue this breeding objective.

Author Contribution

E.W.T. and D.A.V.S. conceived and designed the experiments; E.W.T. performed the experiments; E.W.T. and D.A.V.S. analyzed the data; E.W.T. wrote the paper.

Acknowledgments

This work was funded by grants from USDA Triticeae Coordinate Agricultural Project, N 59-0206-4-002 and the U.S. Department of Agriculture, through the US Wheat and Barley Scab Initiative under Agreement No. 59-0206-9-054. We thank Kathleen Russell, John Connelley and Sandy Swanson for technical assistance.

Conflict of Interest

The authors declare no conflict of interest. The funding sponsors had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript and in the decision to publish the results.

Table 2.1. Means of disease traits for the 2016–2017 study of 238 soft red winter wheat lines grown in a control and warmed treatments, Lexington, KY USA. Below the means, mean squares and level of significance for treatment (T), genotype (G), treatment × genotype (T x G), year, year x genotype (Y x G) and year x treatment (Y x T) are shown for each trait evaluated.

	HD[1]	Rating[2]	PH[3]	Yield[4]	DON[5]	FDK[6]	LBR[7]	PMR[8]	LRR[9]
Control 2016	122.67 A	1.50 A	72.83 A	215.00 B					
Warmed 2016	119.14 B	1.74 A	73.56 A	260.00 A					
Control 2017	114.21 A	1.76 B	89.62 A	572.20 A	4.01 B	11.66 B	3.18 B	1.75 B	2.36 A
Warmed 2017	110.80 B	2.31 A	83.88 B	537.80 B	7.39 A	26.98 A	3.73 A	2.13 A	2.35 A
Treatment (T)	5657.35 **	60.38 ns	2901.21 ns	18.84 ns	2678.49 *	54,941.38 **	73.77 **	35.37 ns	0.004 ns
Genotype (G)	26.02 **	2.54 **	261.59 **	91.06 **	31.84 **	289.86 **	1.12 **	3.02 **	4.02 **
Year	33,292.43 **	61.16 **	86,639.61 **	1,119,898.21 **					
T × G	2.55 ns	1.17 ns	22.11 ns	28.50 ns	10.56 **	84.42 **	0.65 *	0.62 ns	1.14 **
Y × G	4.92 ns	2.02 ns	30.55 ns	44.35 ns					
Y × T	1.39 ns	18.89 **	4904.21 **	1704.82 **					
CV [10]	1.53	56.40	6.10	26.08	39.11	28.85	21.49	44.08	40.24

[1]HD = heading date; [2]Rating = Fusarium head blight rating (1 to 9); [3]PH = plant height (cm); [4]yield (g m−2); [5]DON = deoxynivalenol (ppm); [6]FDK = fusarium damaged kernel (%); [7]LBR = leaf blotch rating (1 to 9); [8]PMR = powdery mildew rating (1 to 9); [9]LRR = leaf rust rating (1 to 9); [10]CV = coefficient of variation; * $p < 0.10$, ** $p < 0.05$, ns not significant

Table 2.2. Pearson correlations among traits for control and warmed treatments in 2016 Lexington, KY. Control treatment above diagonal, warmed treatment below diagonal.

		Control Treatment			
		HD[1]	Rating[2]	PH[3]	Yield[4]
Warmed Treatment	HD		0.02^{ns}	0.19^{**}	-0.09^{ns}
	Rating	0.11^{*}		-0.20^{**}	-0.25^{**}
	PH	0.27^{**}	-0.28^{**}		0.53^{**}
	Yield	0.04^{ns}	-0.28^{**}	0.56^{**}	

[1]HD = heading date (Julian); [2]Rating = Fusarium head blight rating (0 to 9); [3]PH = plant height (cm); [4]yield (g); *, **, ^{ns} significant at $p \leq 0.05$, $p \leq 0.01$ and not significant, respectively.

Table 2.3. Pearson correlations among traits for control and warmed treatments in 2017 Lexington, KY. Control treatment above diagonal, warmed treatment below diagonal.

		Control Treatment								
		HD[1]	Rating[2]	PH[3]	Yield[4]	FDK[5]	DON[6]	LBR[7]	PMR[8]	LRR[9]
Warmed Treatment	HD		-0.02^{ns}	0.35^{**}	0.11^{*}	0.03^{ns}	0.18^{**}	-0.10^{*}	0.07^{ns}	0.26^{**}
	Rating	0.51^{**}		-0.30^{**}	-0.18^{**}	0.40^{**}	0.43^{**}	0.18^{**}	0.04^{ns}	0.10^{*}
	PH	0.47^{**}	0.07^{ns}		0.46^{**}	-0.32^{**}	-0.16^{**}	0.10^{*}	0.14^{**}	0.27^{**}
	Yield	0.10^{*}	0.01^{ns}	0.47^{**}		-0.28^{**}	-0.15^{**}	0.01^{ns}	0.06^{ns}	0.08^{ns}
	FDK	0.01^{ns}	0.38^{**}	-0.26^{**}	-0.22^{**}		0.42^{**}	-0.05^{ns}	-0.02^{ns}	-0.01^{ns}
	DON	0.11^{*}	0.35^{**}	-0.13^{**}	-0.20^{**}	0.57^{**}		-0.03^{ns}	-0.01^{ns}	0.06^{ns}
	LBR	-0.01^{ns}	0.17^{**}	0.13^{**}	0.10^{*}	0.02^{ns}	0.07^{ns}		0.08^{ns}	0.36^{**}
	PMR	-0.01^{ns}	-0.01^{ns}	0.16^{**}	0.09^{ns}	-0.12^{**}	-0.07^{ns}	0.14^{**}		0.05^{ns}
	LRR	0.39^{**}	0.26^{**}	0.39^{**}	0.10^{*}	0.03^{ns}	0.08^{ns}	0.25^{**}	0.03^{ns}	

[1]HD = heading date (Julian); [2]Rating = Fusarium head blight rating (0 to 9); [3]PH = plant height (cm); [4]Yield (g); [5]FDK = fusarium damaged kernels (%); [6]DON = deoxynivalenol (ppm); [7]LBR = leaf blotch rating (1 to 9); [8]PMR = powdery mildew rating (1 to 9); [9]LRR = leaf rust rating (1 to 9); *, **, ^{ns} significant at $p \leq 0.05$, $p \leq 0.01$ and not significant, respectively.

Table 2.4. Broad sense heritability (h^2) and 90% confidence interval (lower limit (LL) and upper limit (UL)) for traits evaluated in control and warmed treatments in 2016 and 2017, Lexington, KY.

	Control Treatment			Warmed Treatment		
Trait	h^2	LL	UP	h^2	LL	UP
HD[1]	0.73	0.67	0.78	0.70	0.63	0.76
Rating[2]	0.23	0.06	0.38	0.13	−0.07	0.29
PH[3]	0.81	0.77	0.85	0.80	0.75	0.84
Yield[4]	0.32	0.17	0.45	0.46	0.33	0.56

[1]HD = heading date (Julian); [2]Rating = Fusarium head blight rating (0 to 9); [3]PH = plant height (cm); [4]yield (g).

Table 2.5. Repeatability and 90% confidence interval (lower limit (LL) and upper limit (UL)) for traits evaluated in control and warmed treatments in 2017, Lexington, KY.

	Control Treatment			Warmed Treatment		
Trait	h^2	LL	UP	h^2	LL	UP
FDK[1]	0.80	0.75	0.84	0.84	0.81	0.87
DON[2]	0.71	0.65	0.77	0.75	0.70	0.80
LBR[3]	0.39	0.24	0.50	0.35	0.20	0.47
PMR[4]	0.42	0.28	0.53	0.69	0.61	0.75
LRR[5]	0.58	0.48	0.65	0.70	0.63	0.75

[1]FDK = Fusarium damaged kernels (%); [2]DON = deoxynivalenol (ppm); [3]LBR = leaf blotch rating (1 to 9); [4]PMR = powdery mildew rating (1 to 9), [5]LRR = leaf rust rating (1 to 9).

Table 2.6. GWAS of 238 soft red winter wheat lines grown in control and warmed treatments in Lexington, KY, 2017. Only SNPs with LOD score >3 are shown. Effect of SNPs is expressed in percent of the mean of each trait.

Trait	Treat[1]	SNP	Chr.[2]	Position	*p* Value	Effect (%)	R^2 (w/o SNP)	R^2 (w/SNP)
FDK[3]	Control	M1563	1B	250.98	0.00033	2.11	0.04558	0.10364
		M2508	2A	173.96	0.00052	−2.32	0.04558	0.09960
		M1591	1B	255.13	0.00059	1.99	0.04558	0.09853
		M6241	3B	285.32	0.00072	−1.82	0.04558	0.09693
		M480	1A	256.00	0.00080	1.72	0.04558	0.09597
		M1905	1B	382.27	0.00087	2.40	0.04558	0.09523
		M8313	5A	311.79	0.00094	−1.73	0.04558	0.09458
	Warm	-	-	-	-	-	-	-
DON[4]	Control	M6959	4A	356.78	0.00018	−0.74	0.12535	0.18343
		M1528	1B	243.59	0.00032	0.87	0.12535	0.17884
		M11423	6B	375.21	0.00034	−0.96	0.12535	0.17827
		M9821	6A	97.50	0.00090	0.63	0.12535	0.17065
		M11046	6B	226.64	0.00094	0.64	0.12535	0.17024
	Warm	M5744	3B	224.48	0.00016	1.13	0.12651	0.18522
		M5743	3B	218.71	0.00074	0.94	0.12651	0.17323
		M7150	4A	564.64	0.00101	0.90	0.12651	0.17085
HD[5]	Control	M13020	7B	433.39	4.84×10^{-5}	0.62	0.05309	0.12743
		M13044	7B	458.56	0.00012	0.56	0.05309	0.11969
		M7239	4A	641.89	0.00018	−0.55	0.05309	0.11587
		M9609	5D	61.48	0.00063	−0.49	0.05309	0.10508
	Warm	M5748	3B	226.99	5.19×10^{-5}	−0.78	0.06517	0.13794
		M5758	3B	231.85	0.00052	−0.55	0.06517	0.11820
		M2132	1D	108.87	0.00065	0.66	0.06517	0.11627
		M12294	7A	456.60	0.00090	0.58	0.06517	0.11353
		M12293	7A	456.60	0.00097	0.57	0.06517	0.11292
		M6708	3D	414.71	0.00104	0.68	0.06517	0.11237

[1]Treat = treatment; [2]Chr. = chromosome; [3]FDK = Fusarium damaged kernels; [4]DON = deoxynivalenol; [5]HD = heading date; [6]PH = plant height.

Table 2.6. Continued.

Trait	Treat[1]	SNP	Chr.[2]	Position	*p* Value	Effect (%)	R^2 (w/o SNP)	R^2 (w/SNP)
PH[6]	Control	M8584	5B	62.86	1.37×10^{-5}	2.41	0.03129	0.11890
		M8577	5B	61.07	0.00051	1.75	0.03129	0.08628
		M9257	5B	357.10	0.00075	2.13	0.03129	0.08302
	Warm	M8584	5B	62.86	6.16×10^{-5}	2.00	0.03537	0.10891

[1]Treat = treatment; [2]Chr. = chromosome; [3]FDK = Fusarium damaged kernels; [4]DON = deoxynivalenol; [5]HD = heading date; [6]PH = plant height.

Table 2.7. Quantitative trait locus (QTL) effect on Fusarium damaged kernels (FDK) and deoxynivalenol (DON) lsmeans of 238 soft red winter wheat lines grown in control and warmed treatments, Lexington, KY, 2017.

QTL	Number of Lines	FDK				DON			
		Control		Warmed		Control		Warmed	
Fhb1–S [1]	218	12.57	B a	27.76	A a	4.60	B b	7.75	A a
Fhb1–R [2]	19	13.08	B a	29.87	A a	6.05	A a	7.18	A a
Rht_B1a	112	13.28	B a	28.31	A a	5.02	B a	8.80	A a
Rht_B1b	123	12.01	B a	27.56	A a	4.49	B a	6.75	A b
Rht_D1a	151	11.94	B b	27.20	A a	4.39	B b	6.69	A b
Rht_D1b	84	13.78	B a	29.18	A a	5.38	B a	9.57	A a
Vrn-A1-short	13	13.39	B a	26.51	A a	3.92	B a	8.67	A a
Vrn-A1	225	12.53	B a	27.99	A a	4.77	B a	7.65	A a
Vrn-B1-short	5	11.39	A a	19.59	A a	3.52	A a	6.33	A a
Vrn-B1	230	12.60	B a	28.00	A a	4.75	B a	7.71	A a
Vrn-D3a-early	70	12.52	B a	28.06	A a	4.38	B a	6.75	A b
Vrn-D3b	165	12.68	B a	27.93	A a	4.89	B a	8.14	A a
Ppd-A1-sensitive	94	12.20	B a	28.65	A a	5.07	B a	8.33	A a
Ppd-A1-insensitive	138	12.92	B a	27.25	A a	4.53	B a	7.23	A b
Ppd-B1-sensitive	175	12.48	B a	27.57	A a	4.77	B a	7.67	A a
Ppd-B1-insensitive	19	13.72	B a	28.73	A a	5.14	B a	8.48	A a
Ppd-D1-sensitive	112	12.36	B a	27.77	A a	4.53	B a	7.34	A a
Ppd-D1-insensitive	123	12.73	B a	27.89	A a	4.85	B a	7.99	A a

Means followed by the same capital letter in the row, for a given trait, do not differ ($p \leq 0.05$) by t test; means followed by the same lower case in the column, for a given treatment, do not differ ($p \leq 0.05$) by t test; [1]*Fhb1*–S = susceptible; [2]*Fhb1*–R = resistant.

Table 2.8. Deoxynivalenol (DON) concentration of soft red winter wheat breeding lines and cultivars in an artificially warmed treatment, Lexington, KY, 2017.

Best Genotypes	Warmed		Control		Worst Genotypes	Warmed		Control	
	DON	Rank	DON	Rank		DON	Rank	DON	Rank
07287RA1-14	1.70	1	1.33	2	07290A1-12	14.18	224	5.05	149
OH08-207-33	1.83	2	1.83	9	MO100172	14.38	225	5.50	167
05287A1-1-13	1.90	3	2.20	23	SS520	14.38	226	4.55	136
MO081652	1.93	4	2.45	33	MO100535	14.58	227	11.90	236
MO081699	2.03	5	1.69	3	OH08-149-11	14.63	228	10.50	229
OH08-101-72	2.08	6	3.25	74	D8006	14.98	229	8.15	219
CLARK	2.33	7	2.25	25	MD03W665-10-3	15.23	230	10.85	233
MO080104	2.38	8	2.37	30	MD03W61-11-2	15.43	231	3.95	109
OH08-234-4	2.38	9	1.78	6	OH07-238-15	15.88	232	7.95	217
IL08-34020	2.58	10	2.00	13	SS5205	17.23	233	11.35	234
KY02C-2215-02	2.58	11	2.75	51	MD03W485-10-2	17.78	234	9.25	226
0570A1-2-39-5	2.58	12	6.05	186	MO100519	18.83	235	8.65	223
0513A1-1-3	2.63	13	2.85	57	03633A1-69-2-5	19.27	236	14.55	237
IL02-19483B	2.73	14	2.55	41	BECKER	19.38	237	3.75	96
IL06-13708	2.78	15	1.90	11	VA10W-140	24.83	238	7.50	210
Average	2.30		2.48		Average	16.74		8.23	

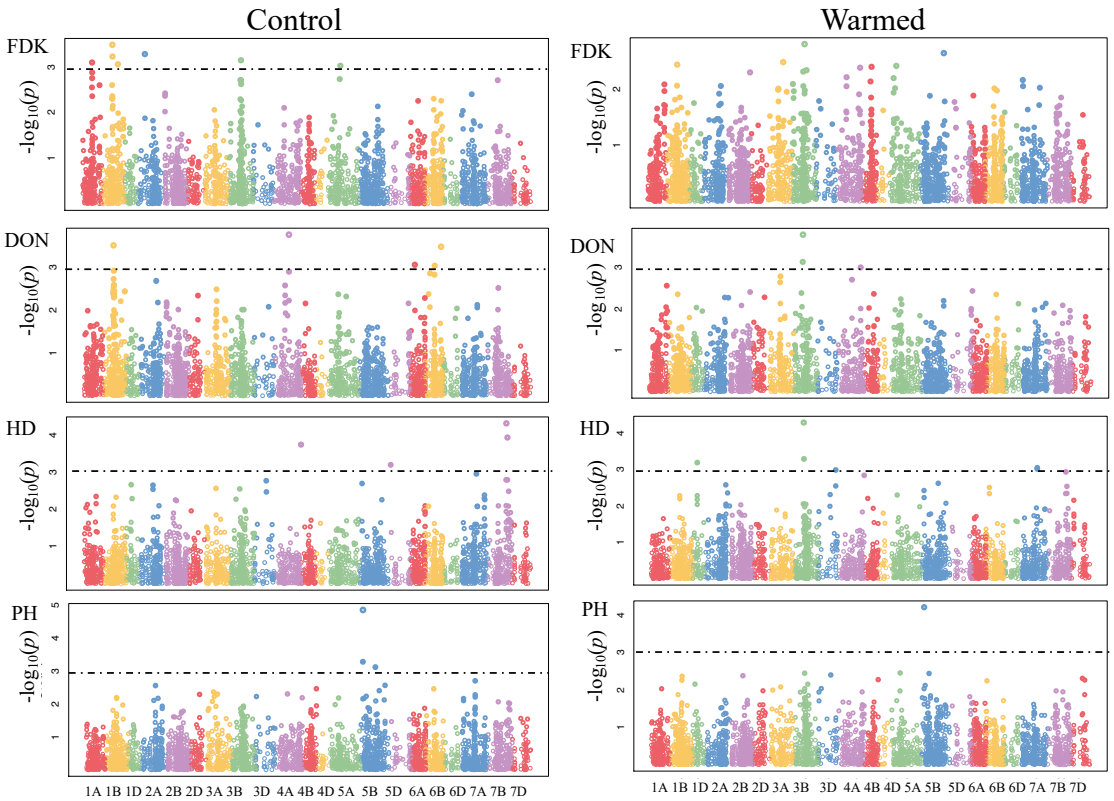


Figure 2.1. Manhattan plots of genome-wide association study (GWAS) was performed for Fusarium damaged kernels (FDK), deoxynivalenol (DON), heading date (HD) and plant height (PH). GWAS results of 238 soft red winter wheat cultivars and breeding lines from the T- CAP panel grown in a control treatment and warmed treatment in Lexington, KY, 2017.

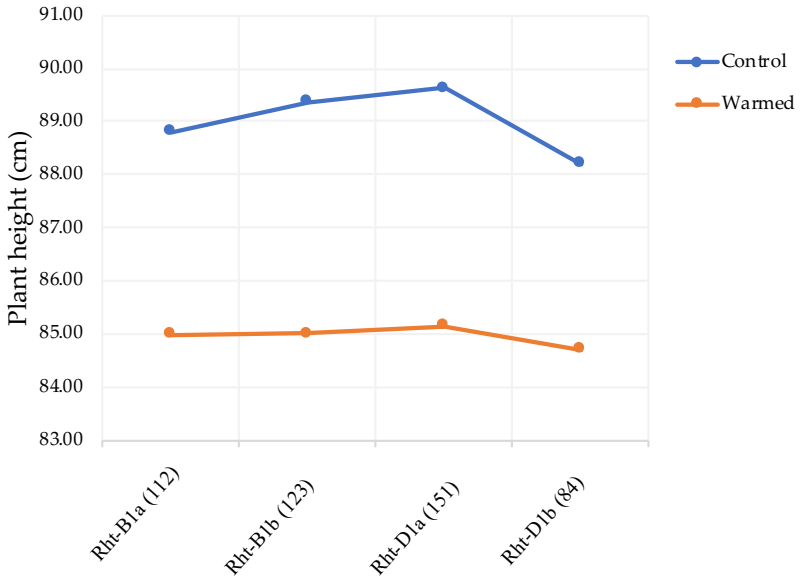
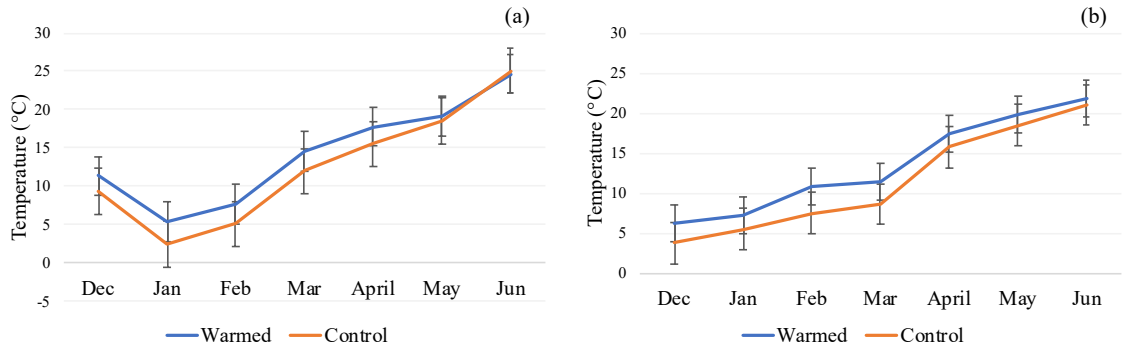


Figure 2.2. Allelic effects of dwarfing genes on plant height in control and warmed treatments, Lexington, KY, 2016-2017. Values in parentheses represent the number of genotypes that have the allele.



Supplemental Figure S2.1. Monthly average of soil temperature in warmed and control treatments from temperature probes placed at a depth of 10 cm below the ground. Temperatures were collected daily every 15 min, 2016 (a) and 2017 (b) at Lexington, KY.

GWAS FOR FUSARIUM HEAD BLIGHT TRAITS IN A SOFT RED WINTER WHEAT MAPPING PANEL

Elisane W. Tessmann[1], Yanhong Dong[2] and David A. Van Sanford[1*]

[1]Department of Plant and Soil Science, University of Kentucky, Lexington, KY 40546, USA;

[2]Department of Plant Pathology, University of Minnesota, St. Paul, MN, 55108

*Correspondence: dvs@uky.edu; Tel.:+1-859-338-2409

Received: 14 August 2018; Accepted for publication in Crop Science

ABSTRACT

Fusarium head blight (FHB) is an important disease of wheat (*Triticum aestivum* L.) that has caused billions of dollars in losses in recent decades. Although a massive breeding effort has been undertaken on multiple continents, there are no wheat cultivars with immunity to the disease. Resistance is conditioned by multiple loci and is further complicated by the role of the environment in expression of the disease phenotype. The objectives of our study were to: (i) evaluate phenotypic response to FHB in a large, diverse soft red winter wheat mapping panel; and (ii) identify promising QTL associated with FHB resistance based on a genome wide association study (GWAS). We evaluated the mapping panel in 2014-2015 and 2015-2016 in an irrigated, inoculated scab nursery near Lexington, KY. Traits evaluated were heading date, plant height, FHB rating, severity, incidence, index, Fusarium damaged kernels (FDK) and deoxynivalenol (DON). There were significant (p<0.05) differences among genotypes for all traits measured. GWAS (based on 2-year entry means) identified 16 significant (p<0.001) SNPs associated with disease traits on multiple chromosomes. SNP association ranged from -2.14 to 4.01% of the mean of a given trait. SNPs associated with FDK and DON were detected on chromosomes 4A, 5B and 6B, and these SNPs decreased DON levels by 2.1, 1.5 and 3.2 ppm, respectively. Our study demonstrated that even small-effect QTL can potentially decrease disease levels and thus be useful in breeding programs.

Introduction

Climate change brings to the agricultural sector the uncertainty of yield production, unpredictable rain patterns, and changes in disease and insect pressure, to name a few challenges. Grain production is estimated to decrease by 6% for wheat (*Triticum aestivum* L.) in the next decades for each increase in degree-Celsius (Zhao et al., 2017). Climate change is also expected to affect occurrence, distribution and intensity of plant diseases such as Fusarium head blight (FHB) (Audenaert et al., 2013). Backhouse et al. (2014) using a modeling approach, found a positive correlation between climate and distribution of pathogenic species of *Fusarium* which includes *Fusarium graminearum*. Their research predicts wide distribution in countries where this disease already occurs and new regions such as Mexico, North Africa, Ethiopia and western Siberia for epidemics of FHB (Backhouse, 2014). Developing genetically improved resistant cultivars in FHB-prone environments will be fundamental to meeting future food demand.

Plant diseases are estimated to cause 10-16% of yield losses globally (Chakraborty and Newton, 2011). Fusarium head blight (FHB) is one of the most important diseases of wheat and other small grains. In the 1990's this disease caused $4.8 billion in losses in the United States (Johnson et al., 2003). During the epidemic years of 1998 through 2000, an estimated $2.7 billion in direct and indirect losses were caused by FHB in wheat and barley (*Hordeum vulagare,* L.) (Nganje et al., 2002).

Fusarium head blight results in yield losses, lowered test weight, reduced percentage of high and low molecular weight glutenins and mycotoxin contamination (Spanic et al., 2017). In the US, FHB is caused primarily by *Fusarium graminearum* a pathogen that likes warm and humid conditions; in cooler, humid environments *F.*

culmorum and *F. avenaceum* also can cause the disease (He et al., 2016a). The disease is capable of threatening farmers and consumers in two ways: first by reducing seed quality and yield through the presence of shriveled "tombstone" kernels that result in low test weight, and second by kernel contamination with mycotoxins (Vaughan et al., 2016; Steiner et al., 2017). Deoxynivalenol (DON) is one of the most important mycotoxins with harmful effects to plants, animal and humans (Audenaert et al., 2013). Levels of this toxin are predicted to increase by a factor of 3 in most regions of north-western Europe due to an earlier flowering period caused by climate change (Fels-Klerx et al., 2012).

FHB is highly influenced by environmental conditions: a susceptible host in warm and humid environment with abundant inoculum during anthesis are the triggers for a severe epidemic (Bai and Shaner, 2004; Jin et al., 2013). Infection occurs during or just after the anthesis, when open florets provide the opportunity for the pathogen to enter and initiate infection (Emrich et al., 2008). FHB is a complex, quantitative disease in which two types of resistance are widely accepted: Type I confers resistance to initial infection, while Type II represents resistance to spread of the pathogen inside the plant (Schroeder and Christensen, 1963). In addition, Mesterhazy et al. (1997) also described three more types of resistance: Type III, resistance to accumulation of toxin; Type IV which is resistance to kernel infection; and Type V, defined as tolerance to the disease.

Breeders in all of the major wheat producing areas have worked hard to develop FHB-resistant cultivars. Resistance is quantitatively inherited with major and minor genes working together. Quantitative trait loci (QTL) such as *Fhb1*, *Qfhs.ifa-5A* and *QFhs.nau-2DL* are widely used in breeding programs as sources of resistance (Kugler et al., 2013; Zhuang et al., 2013; Clark, et al., 2016). Despite these extensive efforts, there is no cultivar

completely resistant to FHB, demonstrating the complexity of this disease. Jin et al. (2013) characterized 363 U.S. winter wheat accessions for FHB resistance; they found that only 7% of the accessions in the greenhouse and 6% in the field showed high levels of resistance. A recent paper by Peterson et al. (2016) reports on a number of QTL present in several soft red winter wheat cultivars. In a recent study published by our group, involving 238 winter wheat genotypes in an artificially warmed environment, we observed that genotypes under warmed conditions had higher levels of disease (Tessmann and Van Sanford, 2018). These are examples that demonstrate the importance of field experimentation to evaluate and select for FHB resistance.

Genome wide association studies (GWAS) have become an important tool to investigate the genetics of traits in large mapping panels. This approach allows one to evaluate the association between each marker and a trait in a large set of lines that are related to varying degrees (Korte and Farlow, 2013; George and Cavanagh, 2015). The advantage of using such a set of lines is to exploit the recombination events that happened over time and contributed to the natural variation observed in the phenotypes (Korte and Farlow, 2013; Huang and Han 2014; George and Cavanagh, 2015; Arruda et al., 2016). Thus, GWAS can be used to identify the genetic architecture of the trait, providing information on the number and the contribution of each locus in the phenotypic response (Korte and Farlow, 2013). Two limitations of GWAS are the difficulty in detecting rare alleles and accounting for population structure (Korte and Farlow, 2013; Huang and Han, 2014; Bazakos et al., 2017). However, GWAS is still a promising method to identify QTL that have an effect across large and diverse population (Jannink, 2007; Ogbonnaya et al., 2017). In this sense, our goal was to identify promising QTL for FHB traits that can provide

information to accelerate and improve selection for FHB resistance. To this end, a two-year experiment was conducted in Lexington, KY, with 256 soft red winter wheat genotypes. Our main objectives were: (i) to evaluate phenotypic response to FHB in a large and diverse soft red winter wheat mapping panel; and (ii) to identify promising QTL associated with FHB resistance based on GWAS analysis.

Materials and Methods

Plant material and field experiments

We evaluated 256 soft red winter wheat cultivars and breeding lines from the Triticeae Coordinated Agricultural Project (TCAP). Funded by the USDA - National Institute for Food and Agriculture, the TCAP project involved 21 states and 55 Universities across the US (http://www.triticeaecap.org/). The TCAP Elite Eastern mapping panel includes cultivars and breeding lines from most of the wheat producing states in the eastern US. A list of entries is shown in supplemental Tables S3.1, S3.2, S3.5 and S3.6.

Our field experiments were planted in the 2014-2015 and 2015-2016 growing seasons. Genotypes were planted in headrows 1.2 m long, spaced 30 cm apart. The planting dates were 23 October 2014 and 19 October 2015 in the FHB nursery at the University of Kentucky's Spindletop Research Farm near Lexington, KY (38°7′37.81″ N, 84°29′44.85″ W). Soil type at the site is a Maury silt loam (fine, mixed, semiactive, mesic Typic Paleudalfs). The experiment was planted in a randomized complete block design with two replications per genotype.

The FHB nursery had an overhead mist irrigation system on an automatic timer that started three weeks before heading. The experiment was inoculated with scabby corn (*Zea mays* L.); inoculum came from 27 isolates taken from diseased seeds collected over the years 2007-2010 from multiple location across Kentucky. The inoculum was prepared by allowing corn to imbibe water overnight before autoclaving. After autoclaving, a solution of 0.2 g streptomycin in 150 mL sterile water was mixed in the corn to avoid the growth of other microorganisms. The corn was inoculated with potato dextrose agar (PDA) containing *Fusarium graminearum*, covered and incubated for 3 weeks until fully colonized by the fungus. After that, the corn was spread on the floor until dry, and put in storage bags in the freezer until use. Approximately 3 weeks prior to heading, the scabby corn was spread in the headrows at a rate of 30 $g.m^{-2}$ (Balut et al., 2013).

Phenotypic evaluation

The following traits related to FHB in winter wheat were evaluated: heading date (HD), FHB rating, incidence (INC), severity (SEV), index, plant height (PH), Fusarium damaged kernels (FDK), and deoxynivalenol (DON).

Heading date was recorded (in Julian days) for each individual plot when more than 50% of the spikes in the row had emerged. Twenty-four days after HD in a given plot, incidence, severity and FHB rating were evaluated. Incidence, which estimates the percentage of the spikes in a plot that are infected by the fungus, was assessed by counting the number of blighted spikes in a random sample of 20 spikes and converting to percentage. Severity estimates the spread of the disease in the spike; for that we counted the number of infected spikelets per total spikelets in 10 blighted heads (expressed as

percentage). Index (%) was obtained by multiplying severity and incidence and multiplying the product by 100. FHB rating was a visual estimate of FHB index ranging from 0 (absence of FHB symptoms) to 9 (≥ 90% of FHB blighted spikelets).

Plant height (cm) was measured from the soil surface to the top of the spike, excluding awns. Lines were manually harvested using a sickle, mechanically threshed and cleaned. After cleaning, a grain sample of approximately 15 g from each row was further cleaned by hand and evaluated for Fusarium damaged kernels (FDK). An air separation machine specifically developed from a Precision Machine head thresher and a Shop-Vac vacuum to separate scabby kernels from healthy ones as described in Agostinelli et al. (2012) was used for FDK. Scabby kernels and healthy kernels were weighed separately and FDK was calculated by:

$$FDK(\%) = \left(\left(\frac{W_{sk}}{W_{sk} + W_{hk}} \right) x\ 100 \right)$$

where W_{sk} = weight scabby kernel (g); and W_{hk} = weight healthy kernel (g). The same sample (15 g) was sent to the University of Minnesota DON testing laboratory for DON analysis. DON concentration was determined by gas chromatography with mass spectrometry (Mirocha et al. 1998; Fuentes et al., 2005).

Phenotypic data analysis

Analysis of variance (ANOVA) was done using the General Linear Models procedure in SAS (Proc GLM; SAS Institute Inc., Version 9.3) to determine the significance of the main effect genotype. The model used was:

$$\gamma_{ijkl} = \mu + S_i + R(S)_{ij} + G_k + Y_l + G_k * Y_l + \varepsilon_{ijkl}$$

Where: Y_{ijkl} = the observation of the *kth* genotype in *jth* rep in the *ith* set in the *lth* year; μ = the overall mean; *Si* = the effect of *ith* set; *R(S)ij* the effect of *ith* set within *jth* rep; G_k = the effect of the *kth* genotype; Y_l = the effect of the *l*th year; $G_k * Y_l$ = the effect of the interaction of the *lth* year and the *kth* genotype, ε_{ijkl} = the residual error. Sets were used here as a blocking device; since the nursery was planted using trays, rows contained within the foot print of a tray consisted a set.

Broad sense heritability of the traits measured in this study was estimated on an entry mean basis using the model above. To obtain the expected mean squares (EMS) and heritability, data was analyzed using Proc Varcomp in SAS (SAS Institute Inc., Version 9.3). The following equation was used to estimate heritability for the traits:

$$h^2 = \frac{\sigma_g^2}{\frac{\sigma^2}{re} + \frac{\sigma_{ge}^2}{e} + \sigma_g^2}$$

Where: h^2= heritability, σ_g^2= genotypic variance, σ^2= error variance; σ_{ge}^2= genotype x environment variance and *r* and *e* denote the number of reps and environments (Fehr, 1987). Confidence intervals (90%) were calculated after Knapp et al. (1985).

Correlations among traits were estimated using entry means in JMP 13.2 (SAS Institute Inc., Version 9.3) for each year of this study.

Genotypic data

Our study involved two hundred and fifty of the entries in the mapping panel. Six of the 256 entries were omitted from the GWAS due to incomplete genotypic information. The 250 lines were genotyped with the 90K Illumina SNP to identify single nucleotide

polymorphisms. The genotyping process was conducted at the USDA-ARS Biosciences Research Laboratory, Fargo, ND, U.S.

The original number of markers was approximately 28000. The lab group of Dr. Clay Sneller at The Ohio State University (Columbus, OH) removed markers with minor allele frequency <10%, missing data >5% and a SNP tagging method was used to define 3919 independent markers (Mao Huang, personal communication, 2017).

Genotyping for height (*Rht-B1* and *Rht-D1)*, vernalization (*Vrn-A1, Vrn-B1* and *Vrn-D3*), and photoperiod (*Ppd-A1, Ppd-B1,* and *Ppd-D1*) QTL was done in the USDA-ARS Eastern Regional Small Grains Genotyping Laboratory in Raleigh, NC (https://www.ars.usda.gov/southeast-area/raleigh-nc/plant-science-research/docs/small-grains-genotyping-laboratory/main/) using KASP (Kompetitive allele specific PCR) markers. Details on KASP markers used in this study were published by Guedira et al. (2016). Mean comparisons between allele of a specific QTL were realized using *t* test in JMP 13.2 (SAS Institute Inc., Version 9.3). For QTL with large differences in number of lines, we performed Welch's *t* test in JMP 13.2 (SAS Institute Inc., Version 9.3).

Genome wide association study

For the GWAS analysis we used the package Genomic Association and Prediction Integrated Tool (GAPIT; Lipka et al., 2012) R package. GAPIT uses a compressed mixed linear model approach for the GWAS to identify SNPs associated with the traits of interest. The model used can be expressed as follows (Yu et al., 2006):

$$Y = X\beta + S\alpha + Qv + Zu + e$$

in which $Y =$ represents the phenotype; β = is an unknown fixed effect vector that contains genetic marker, population structure and intercept; α = is a vector of SNP effects; v = is a vector of population effects; u = unknown vector of random additive genetic effects from multiple background QTL for individuals or lines; X, S, Q and Z = are known matrices; e = residual. Heading date and the QTL *Rht-B1, Rht-D1, Vrn-A1, Vrn-B1, Vrn-D3, Ppd-A1, Ppd-B1 and Ppd-D1* were used as covariates in the model. We used all QTL together as covariates for all traits analyzed in this study.

We used TASSEL (http://www.maizegenetics.net, Bradbury et al., 2007) to analyze population structure; there was no underlying structure in the data set that was identified in the analysis. Similarly, the principal components analysis in GAPIT did not reveal a structural pattern in the data. QQ plots for all traits are presented in supplemental Fig. S3.1, S3.2 and S3.3. QQ plots showed that the observed *P* value corresponded to the expected values for each trait. There is no early separation of the expected from the observed *P* value, which would indicate population stratification (Ehret, 2010).

We used the information from GAPIT on the three SNPs associated with FDK and DON to ascertain the impact of the disease reducing alleles in lines that contained them at each SNP. By comparing means of lines whose SNP genotype differed – AA vs TT, and using a t-test to assess the significance of the difference, we were able to determine the robustness of the SNP effect across a diverse array of lines (Table 3.5, S3.2).

Results

Phenotypic analysis

To assess genetic variability among the genotypes in this study, we performed means comparisons and analysis of variance for eight scab traits. Significant differences among genotypes ($p<0.05$) were observed for all traits considered in this study (Table 3.1). Additionally, there were significant differences between years ($p<0.05$) for all traits as well as significant ($p<0.05$) genotype x year interaction. Heading date, on average, was 6.4 days earlier in 2016 than in 2015. Plant height differed by 4 cm between years ($p<0.01$); in 2015 average plant height was 87.7 cm versus 83.7 cm in 2016.

Disease levels were higher in 2015 than in 2016 for all traits with the exception of DON. FHB rating was 44.1% lower in 2016 than in 2015: 6.8 vs 3.8 in 2015 and 2016, respectively. Severity was 39.7% lower in 2016 than in 2015; similarly, incidence was slightly lower in 2016 than in 2015 by 4.1% and FHB Index was lower in 2016 than in 2015 by 45.1%. FDK followed the same trend: the 2016 mean was 23.2% lower than that of 2015. Somewhat surprisingly, DON levels in 2016 exceeded the 2015 average of 8.9 ppm by 48.3%. Histograms showing trait distributions in single years and from the combined analysis are presented in supplemental Fig. S3.4, S3.5 and S3.6.

We estimated heritability of the scab traits based on 2-year entry means (Table 3.1). As expected, HD and PH had high heritability estimates: 0.64 and 0.79 respectively. Heritability of FHB rating was 0.60, whereas severity, incidence and index had low heritabilities, with values of 0.31, 0.20 and 0.28, respectively. Heritability estimates of FDK and DON were 0.69 and 0.77, respectively.

Correlations among traits evaluated in 2015 and 2016 are shown in Table 3.2. Heading date was significantly positively correlated with all FHB traits in 2015 except for Rating and FDK where the correlations were non-significant. In 2016, however, a different pattern prevailed: the correlations between HD and scab traits were mostly significantly negative with the exception of INC and DON, which had low positive correlations. Plant height had a significant low to moderate negative correlation with all FHB traits in 2015, with the exception of HD, (r = 0.26). In 2016, PH was significantly negatively correlated with most traits, the exceptions being a positive correlation of 0.51 with HD and no correlation with incidence.

FHB rating is a visual estimation that reflects the spread of disease in the plot (incidence) and in the spike (severity). FHB rating was significantly correlated with severity and incidence in both years of the study, with values ranging from 0.11 – 0.73. FHB rating and FDK had moderately positive correlations in both years, with values of 0.50 and 0.56 in 2015 and 2016, respectively, and FHB rating and DON were also positively correlated in 2015 and 2016 with respective values of 0.43 and 0.35.

Severity had moderate, significant positive correlations with FDK and DON in both years. Positive correlations of 0.71 and 0.59 were observed between FDK and DON in 2015 and 2016, respectively (Table 3.2).

Genome wide association study (GWAS)

We performed GWAS to identify promising QTL associated with FHB resistance that might improve selection for resistance. QTL such as *Rht-B1a, Rht-B1b, Rht-D1a, Rht-D1b, Vrn-A1, Vrn-B1, Vrn-D3, Ppd-A1a, Ppd-A1b, Ppd-B1a, Ppd-B1b, Ppd-D1a and Ppd-*

D1b were used as covariates in the model. Since genotypes in the panel were from different latitudes, and thus differed for HD, we also used HD as a covariate in the model. The GWAS analysis was conducted using 2-year entry means as well as individual year entry means. SNPs and the magnitude of their effects are presented in Table 3.3; only SNPs with LOD scores > 3 are listed in the table.

In the 2-year GWAS, a total of 16 SNPs were detected across the traits in this study (Fig. 3.1), five of which had disease reducing effects. These five SNPs were located on chromosomes 4A, 5B, 6B and 7A (Fig. 3.1, Table 3.3, S3.3). SNP effects as a percent of the trait mean ranged from -2.14% for plant height to 4.01% for incidence. The association mapping for PH identified one SNP located on chromosome 6A with an effect of -2.14%. There were three SNPs on chromosomes 7A and 7B associated with rating; the two on chromosome 7B, M12955 and M12960, had effects of 0.39 and 0.35%, respectively. There were no SNPs associated with severity that were revealed by the analysis.

GWAS for incidence, on the other hand, identified 5 SNPs on chromosomes 7B and 3A. The three SNPs on chromosome 7B, M12955, M12960, and M12957 had respective effects of 4.01, 3.76 and 3.10%, while SNPs M5559 and M5589 on chromosome 3A, had effects of 2.86 and 2.63%, respectively.

The three SNPs on 7B associated with incidence were also identified for FHB index, with effects ranging from 2.60 to 3.05%. There were two SNPs (M9432 and M11432) on chromosomes 5B and 6B associated with FDK; with respective effects of -0.64 and -0.86%. SNP M9432 (5B) and M6959 (4A), were associated with DON, with estimated effects of -1.42 and -1.17%, respectively (Table 3.3, Fig. 3.1).

In addition to the GWAS using least squares means over the two years, we also performed GWAS for individual years (Table 3.3). Manhattan plots for the single years are provided as supplementary material (Fig. S3.7 and S3.8). It is interesting that there were no SNPs associated with PH, rating and DON in 2015. In addition, five SNPs were associated with SEV, explaining from -0.50 to -5.04% of the mean for the trait. Two SNPs were associated with INC on chromosomes 2B and 7B with effects of -3.91 and 5.02%, respectively in 2015. SNPs associated with FDK showed effects of -1.05 and -1.09% for chromosomes 2A and 5B in 2015, respectively.

In 2016, the GWAS highlighted a total of 16 SNPs associated with the traits in this study. Four SNPs on chromosomes 1B and 5A were associated with rating in 2016, with effects ranging from -0.37 to 0.38%. These results differ from the 2-year GWAS where SNPs on chromosomes 7A and 7B were associated with rating. Similar to 2015, four SNPs associated with SEV in 2016 had effects ranging from -2.51 to 1.37%. Two small-effect SNPs were associated with FDK in 2016, explaining 0.63 and 0.78% of the variation observed. For DON, SNPs on chromosomes 7D and 5B with effects of 2.53 and -1.47%, respectively, were detected in 2016.

The impact of known QTL on the genotypic response to FHB rating, FDK and DON are presented in Table 3.4. The QTL analyzed in the population were: *Fhb1*, height (*Rht-B1* and *Rht-D1*), vernalization (*Vrn-A1, Vrn-B1* and *Vrn-D3*) and photoperiod (*Ppd-A1, Ppd-B1 and Ppd-D1*) genes. These QTL were chosen for analysis because they are either important FHB resistant genes (*Fhb1)* or fundamental growth and development genes that have been implicated in FHB studies (e.g., *Ppd-D1*; Islam et al., 2016). Using the average of each trait, the population was classified for each allelic form, and levels of

PH, HD, FHB rating, FDK, and DON were calculated for each QTL. The remaining traits and a complete QTL description for each line are in the supplemental material (Tables S3.4, S3.5 and S3.6). It would have been desirable to test for the presence of resistance alleles at the newly identified QTL from 'Bess' and 'Neuse' reported in Peterson et al. (2016). However, when the TCAP population was genotyped in 2013, these markers were not available (Gina Brown-Guedira, personal communication, 2019).

Two hundred and twenty-nine genotypes did not have resistance alleles at *Fhb1* while only nineteen panel entries did. There was no significant difference between the group of genotypes with *Fhb1* – S alleles and the group with *Fhb1* – R alleles for the traits evaluated.

In the mapping panel, lines with the dwarfing allele *Rht-B1b*, on average, showed 7.3 and 16.5% lower FHB rating and DON respectively, than lines with the wild type alleles. Surprisingly, no differences were found between the wild type and dwarfing alleles for FDK. The dwarfing allele *Rht-D1b* was associated with 9.8 and 26.5% more disease than the wild type allele (*Rht-D1a*) for FHB rating and DON, respectively, though no difference between dwarf and wild type alleles was observed for FDK. No differences were observed for plant height for any of the allelic forms of *Rht-B1* and *Rht-D1*. Interestingly in the case of HD, a slight increase (0.6%) was observed for *Rht-D1b* in comparison to *Rht-D1a* (Table 3.4).

We also analyzed vernalization and photoperiod alleles and their relationship to FHB traits. Significant differences were observed for *Vrn-B1* and *Vrn-D3*; a decrease in DON of 21.8% for *Vrn-B1-short*, while for *Vrn-D3a-early* 9.3 and 14.7% for rating and DON was observed. For photoperiod alleles, *Ppd-A1* showed significant differences for

HD and DON with respective increases of 1.0 and 14.4% for *Ppd-A1b*. Significant differences were observed for all traits in relation to *Ppd-D1*. Respective increases of 3.3 and 0.6% were observed for PH and HD when Ppd-D1b was present. Decrease in rating, FDK and DON were observed for *Ppd-D1b*; 9.1, 10.9 and 12.7%, respectively.

Discussion

GWAS study

Developing new cultivars capable of maintaining or increasing yield production under biotic and abiotic stresses is a major challenge for food production in this century. Diseases such as FHB can drastically reduce production and affect grain quality, resulting in low revenue for farmers. Field experiments such as this one, in which a large diverse panel is used, can facilitate identification of potential QTLs associated with FHB resistance.

GWAS results differed between years and individual years differed from results based on the 2-year means (Table 3.3). SNPs were associated with SEV in each year, however the 2-year GWAS did not identify any significant SNPs associated with SEV. A possible explanation could reside in the environmental conditions which differed greatly between years and thus affected the phenotypic response of panel entries (Table 3.1, Fig. S3.9). For example, a difference of 39.7% was observed between years for SEV. A majority of the SNPs identified in the individual year GWAS decreased SEV both years and could be a useful tool in selection for resistance, though their absence in the 2-year GWAS is not encouraging. Petersen et al. (2016) identified resistant QTL on chromosomes 1A, 2A and

6A associated with SEV in the winter wheat NC-Neuse. Liu et al. (2013) identified two QTL on chromosomes 1D and 3B that decreased SEV. In our study, GWAS detected SNPs on chromosomes 1A, 1B, 1D, 3A and 5B for 2015 and 4A, 6B and 7B for 2016 (Table 3.3, Fig. S3.7 and 3.8).

From the GWAS using 2-year means, three SNPs (M12955, M12960 and M12957) on chromosome 7B increased rating, INC and Index. The SNPs explained from 0.35 to 4.01% of the phenotypic variation for those traits (Table 3.3, Fig. 3.1). The SNP M12960 was the only one detected in 2016 GWAS, and it also increased disease with an effect of 5.02%. Gilsinger et al. (2005), studying flower opening in recombinant inbred lines, identified chromosome 7B to be associated with low incidence. Two SNPs on chromosome 3A were associated with increased INC in the combined analysis. It is interesting that in the 2015 analysis the SNPs detected on chromosome 3A decreased FHB severity and index levels. Steiner et al. (2004) found chromosome 3A to be associated with a reduction in severity and incidence in the cultivar Frontana, suggesting that SNPs on this chromosome could be useful in breeding programs. We only observed decreased severity and incidence in 2015; not in 2016 or in the 2-year analysis.

FDK and DON can have an impact on grain commerce. Thus, SNPs associated with FDK and DON would be very useful to wheat breeders. FHB resistance QTL have been identified on all 21 chromosomes (Buerstmayr et al., 2009; Liu et al., 2009; Cai et al., 2016). The cultivar Truman was studied by Islam et al. (2016) and QTLs associated with FDK and DON were found on chromosomes 2A, 2D and 3B. Our GWAS for FDK and DON identified SNPs on chromosomes 5B, 6B and 4A. SNP M9432 on chromosome 5B was associated with reduced FDK and DON; it explained 0.64 and 1.42% of the phenotypic

variation in the two traits respectively. Similarly, SNP M11423 on chromosome 6B decreased FDK 0.86%, while SNP M6959 on chromosome 4A reduced DON 1.17%. Bonin and Kolb (2009) also reported that chromosome 6B was associated with kernel damage; and Liu et al. (2009) identified chromosomes 5B and 6B as important for FHB resistance breeding. In addition, Peterson et al (2016) identified chromosomes 4A and 5B; and Liu et al. (2012) observed chromosome 5B to be associated with DON.

In order to assess impact on disease levels associated with SNPs on chromosomes 5B, 6B and 4A, we identified lines with alternate alleles at those SNPs. After classifying the lines for each allelic form of the SNP genotype, we used phenotypic data to assess disease levels (Table 3.5). Although SNPs presented in Table 3.5 were associated with FDK and DON, we were also interested in the effects of these SNPs on the other scab traits measured in this study. Lines with the TT genotype at SNP M9432 showed a decrease of 17.4% in FDK when compared with lines with the AA genotype. No differences in FDK were observed for SNP M6959. Similar to the SNP M9432, the TT genotype at SNP M11423 decreased FDK levels by 18.1%.

Significant differences (p<0.05) in DON levels between SNP genotypes were observed for all three SNPs. Lines with the TT genotype at the SNPs M9432 and M6959 had DON levels that were 16.8 and 12.3% lower than lines with the AA genotype, respectively. Though SNP M11423 was only associated with FDK in the GAPIT; TT genotypes at this SNP had DON levels 3.2 ppm lower than AA genotypes, a 23% reduction. Thus, even though the magnitude of SNP effects was small when expressed as a percentage of the mean: -1.42 and -1.17% for M9432 and M6959 and -0.64 and -0.86% for M9432 and M11423, respectively, the presence of the disease reducing alleles at these SNPs

significantly lowered DON levels. Lines with the TT genotype at SNP M11423 showed decreased severity, incidence and index by 10.7, 6.6 and 15.7%, respectively. This SNP also lowered FDK slightly (-0.86 %) and showed a decrease in disease levels for all FHB traits with the exception of FHB rating. Based on these results, SNP M11423, could be useful in FHB resistance breeding.

In our study, the magnitude of SNP effects was small, ranging from -2.14% to 4.01% of the mean of the traits measured (Table 3.3). The small effects observed here are consistent with the complexity of FHB. This disease is highly affected by genotype-by-environment interaction and major and minor genes are involved in conferring resistance (Buerstmayr et al., 2009; Miedaner and Korzun, 2012; Cai et al., 2016). An increase in trait complexity decreases the likelihood of detecting large effect QTL; it is more likely that one will detect small QTL effects when multiple genes are involved in the trait (Robertson, 1967; Massman et al., 2011). Major QTL are most likely fixed in the population due to strong selection during multiple years for the desired QTL (Massman et al., 2011). Although the effects detected in this study were small, the respective differences in DON levels associated with SNPs M9432, M6959, and M11423 - 2.1, 1.5 and 3.2 ppm, are meaningful, given the FDA advisory limit of 1 ppm on grain sold for human consumption.

Selection of lines to be used in breeding programs can be optimized by understanding the genetic architecture of the trait, allowing breeders to better determine which elite lines to use in crosses (Massman et al., 2011; Xiao et al., 2017). GWAS provides information such as the number of genes involved in controlling a trait and the effects of the SNP (Schmid and Bennewitz, 2017). GWAS can also be used to inform genomic selection (GS) models, where highly significant SNPs revealed by the GWAS can

be used as fixed effects in the GS model (Begum et al., 2015). In the present study, even though SNP effects were small, FDK and DON showed meaningful reductions, and thus could be used in a GS program or when devising crosses to increase FHB resistance via marker assisted selection (Massman et al., 2011).

QTL for FHB Rating, FDK and DON

In addition to the GWAS analysis, we were also interested in evaluating the possible role of important QTLs in affecting FHB rating, FDK and DON levels (Table 3.4). One of the most used resistance QTLs is *Fhb1* (Rawat et al., 2016; Steiner et al., 2017). In the population from our study, 229 lines did not have resistance alleles at *Fhb1* while only 19 did. There was no significant difference between *Fhb1*-S and *Fhb1*-R for FHB rating, FDK and DON, although the magnitude of the ranges differed between the two groups. DON, for example in the *Fhb1*-R group ranged from 6.1 to 14.8 ppm; in the *Fhb1*-S group, the range was much larger: 3.7 to 33.3 ppm (data not shown).

Plants with *Rht-B1b* showed more resistance in terms of FDK and DON, while plants with *Rht-D1b* had increased FHB ratings and DON levels. Klahr et al. (2007) observed a negative correlation between plant height and FHB; they suggested that short statured genotypes are more susceptible to FHB due to the spike's proximity to the inoculum and a microenvironment with high moisture and humidity around the spike. On the other hand, Hilton et al. (1999), analyzing relative humidity at ear height in isogenic lines for *Rht-B1* and *Rht-D1* observed no differences between short and tall genotypes. These authors suggested that microclimate around the spike could not explain the FHB severity in those lines (Hilton et al., 1999). Srinivasachary et al. (2009) showed that the

presence of the alleles *Rht-B1b* and *Rht-D1b* decreased resistance to initial infection and, while *Rht-B1b* increased Type II resistance, *Rht-D1b* had no effect on it. Many studies suggest the negative effects of *Rht-B1b* and *Rht-D1b* in resistance to FHB (Srinivasachary et al., 2008; Mao et al., 2010; Kollers et al., 2013; Lu et al., 2013; Buerstmayr and Buerstmayr, 2016). As shown in this study, and that of Lu et al. (2013), plants with the dwarfing allele *Rht-B1b* had less disease; on that basis, this allele is more favorable for use in FHB resistance breeding programs.

Vernalization response and photoperiod sensitivity genes are responsible for controlling flowering during favorable conditions, ensuring wide adaptability of wheat (Gomez et al., 2014; Guedira et al., 2016). For vernalization genes, no differences were observed for *Vrn-A1* for FHB rating, FDK, and DON (Table 3.4). Differences were observed for *Vrn-B1* and Vrn-*D3b*; *where Vrn-B1-short* and *Vrn-D3a-early* genotypes had a decrease in DON levels. For photoperiod genes, a decrease in DON levels was observed for *Ppd-A1a* genotypes, while for *Ppd-D1a* an increase in FHB rating and DON were observed (Table 3.4). Similar results were observed by Koller et al. (2013) who identified increased FHB severity for *Ppd-D1a* genotypes when compared with photoperiod sensitive which had increased resistance to FHB via lower severity.

Phenotypic characteristics

Genetic variability is the basis for the development of new varieties with high yields, agronomic fitness, disease resistance and adaptability to different environments. Evaluation of genotypes under field conditions is fundamental for assessing plant response to environmental stresses, such as disease pressure. Through FHB resistance is determined

by resistance genes, heading date can play an important role in determining the amount of disease. Positive correlations between HD and FDK and DON have been observed in some studies, suggesting that early HD provided escape of the optimal infection window (Petersen et al., 2016; Liu et al., 2013). In our study, significant differences were observed for HD in both years, and genotypes were ~ 6 days earlier in 2016 (Table 3.1). Disease levels were lower in 2016 with the exception of DON which had higher levels than were recorded in 2015.

Heading date was positively correlated with disease traits in 2015, however we did not observe the same trend in 2016 when FHB rating, severity, index, and FDK were negatively correlated with HD (Table 3.2). Our results in 2015 suggested that genotypes with an early heading date had lower disease levels; similar results have been reported in the literature (Petersen et al.,2016; Liu et al., 2013). However, in 2016 early genotypes seemed to have more disease than later heading genotypes.

As discussed previously, plant height can affect disease pressure (Buerstmayr et al., 2002; Mao et al., 2010; Buerstmayr and Buerstmayr 2016; Schulthess et al., 2018). Correlations between PH and FHB traits were negative in both years of our study, which agrees with the literature (Mao et al., 2010; Yan, et al., 2011; Buerstmayr and Buerstmayr 2016). Average PH was 4 cm less in 2016 than in 2015, though disease trait values, with the exception of DON, were lower in 2016 (Table 3.1). A possible explanation for the variation in disease levels might be found in the temperature difference between 2015 and 2016. Temperatures during 2016 were higher than in 2014-2015 (Fig. S3.9). During February and March, increases of 7° and 4°C, respectively, were observed in 2016 when compared with 2015. Plants in 2016 flowered at the beginning of May, in contrast to 2015

when flowering began in the middle of May. A difference in May temperatures of 2.4°C was observed between years. A cooler environment during fungal colonization in 2016 probably slowed down its development so that spike symptoms were less visible, even though DON levels in 2016 ultimately turned out to be higher (Table 3.1).

Among the FHB traits evaluated in the field, rating has an advantage: assessment is rapid and less labor is required. To rely only on FHB rating in the field to evaluate disease, however, requires that the trait has high heritability and a strong correlation with other measures of FHB. In both years of this study, FHB rating was positively correlated with severity and incidence (Table 3.2). In addition, FHB rating also had a moderate correlation with FDK and DON both years. A moderately high heritability of FHB rating was estimated (0.6), over the two years of the study (Table 3.1). Based on these findings, FHB rating could be used instead of severity and incidence to evaluate FHB spike symptoms.

FDK was estimated using air separation of sound and scabby kernels and by a visual disease estimation. The advantage of this method is the low cost of the analysis; however, the visual estimation is dependent on the person evaluating the sample. To use FDK as a selection method for FHB resistance, a high correlation between FDK and DON is necessary. We observed a correlation of 0.71 (p<0.01) was observed between FDK and DON in 2015, and 0.59 (p<0.01) in 2016 (Table 3.2). Heritability estimates for FDK and DON were, 0.69 and 0.77, respectively, indicating likely progress from selection.

The 20 mapping panel entries with the highest DON concentrations and the 20 with the lowest DON concentrations are shown in Tables 3.6 and 3.7, respectively. We were interested to know how these lines could be characterized for plant height, heading date

and to ascertain their genotypes at the *Rht*, *Vrn*, *Ppd* and *Fhb1* loci. Average DON levels were 5.0 ppm for the lowest DON lines and 21.6 ppm for the highest DON lines. Toxin levels ranged from 3.7 to 5.7 ppm in the low DON group, and from 18.2 to 33.3 ppm in the 20 high DON group.

As expected, lines in the low DON group were taller when compared with the high DON group: 87.7 vs. 81.5 cm. Heading date differed by 1 day between low and high DON groups, with average values of ~ 126 vs ~127 days, respectively. The range among the lines in each group casts doubt on the importance of this 1 day difference, however: in the lowest DON lines, HD ranged from 123 to 133.3 days, while in the highest DON lines the range was 123 to 130.5 days. An overall look at *Rht* alleles in the lowest DON lines showed that 12 genotypes had the dwarfing allele at *Rht-B1*, while only 3 genotypes had the dwarfing allele at *Rht-D1*. The opposite scenario was observed among the highest DON lines, where only 2 genotypes had *Rht-B1b* and 16 genotypes had *Rht-D1b*. Strong evidence of the association of the *Rht-D1* wild type allele and decreased resistance to initial infection is reported in the literature (Draeger et al., 2007; Srinivasachary et al., 2008, 2009; Lu et al., 2011). Our results agree with this finding for *Rht-D1b*, in that almost all of the highest DON concentration lines had this allele at *Rht-D1*. In addition, the lowest DON lines had the wild type allele for *Rht-D1*. Caution is advised, however, in drawing inferences from just the extreme subsets of the panel.

For vernalization genes, a majority of the 20 genotypes in each DON group had *Vrn-A1* and *Vrn-B1*. A different scenario was observed for *Vrn-D3*, where 11 low DON genotypes did not have this allele; while 16 of 20 high DON genotypes had the mutant

allele. A look at the overall population showed a similar trend where lines with *Vrn-D3* had more disease based on FHB rating and DON (Table 3.4).

For *Ppd-A1*, 13 genotypes in the low DON group were classified as *Ppd-A1a*, while 5 genotypes were *Ppd-A1a* in the high DON group. The overall population showed significantly (p<0.05) higher levels of DON for *Ppd-A1* sensitive genotypes (Table 3.4). Similar results were observed in the high DON group, where most genotypes were *Ppd-A1b*. For *Ppd-B1*, the majority of the low DON lines had the sensitive type; as did 13 of the high DON genotypes. Similarly, the population as a whole did not show differences between *Ppd-B1b* and insensitive for disease traits. Half of the genotypes in the lowest DON lines were classified as *Ppd-D1a*; for the highest DON group, 16 genotypes were classified as *Ppd-D1a* (Table 3.7). The overall population had similar results, where *Ppd-D1a* genotypes had higher FHB rating and DON levels (Table 3.4).

None of the genotypes classified as lowest and highest DON concentration had the resistance alleles at the *Fhb1* locus. With regard to plant height, it is interesting that the entry IL01-11934, with very low DON, was shorter than the average height of lines in either DON group. Plants of similar height in the high DON class had much higher levels of DON, e.g. 19.7 ppm for VA10W-21 and 33.3 ppm for Crystal (Table 3.7). Based on a companion study (Tessmann, unpublished) we know that IL01-11934 has morphological traits associated with reduced scab.

Conclusion

In this study, 16 SNPs in the 2-year GWAS were associated with FHB traits. SNPs with disease reducing effects for FDK and DON were identified on chromosomes 5B (M9432), 6B (M11423) and 4A (M6959). A significant (p<0.05) decrease in DON levels was observed for lines with the TT versus the AA genotype at these SNPs. Our study demonstrated that even a small effect QTL can potentially be valuable in breeding programs.

Conflict of Interest

The authors declare that there is no conflict of interest.

Acknowledgments

This work was funded by grants from USDA Triticeae Coordinated Agricultural Project, N 59-0206-4-002 and the U.S. Department of Agriculture, through the US Wheat and Barley Scab Initiative under Agreement No. 59-0206-9-054. We thank Dr. Anthony Clark, John Connelley and Sandy Swanson for technical assistance; Dr. Gina Brown-Guedira and her lab for running KASP assays on major QTLs and Dr. Clay Sneller and his lab for providing a curated set of genomic markers.

Author Contributions

E.W.T. and D.A.V.S. conceived and designed the experiments; E.W.T. performed the experiments; Y. D. ran the DON analysis; E.W.T. and D.A.V.S. analyzed the data; E.W.T. wrote the paper.

Table 3.1. Means of scab traits for the 2015-2016 study of 256 soft red winter wheat lines grown in Lexington, KY. Below the means, mean squares and level of significance for genotype, year, and genotype x year (G x Y) and broad sense heritability (h^2) and 90% confidence interval (lower limit (LL) and upper limit (UP) are shown for each trait.

Traits	HD[1]	PH[2]	RATING[3]	SEV[4]	INC[5]	INDEX[6]	FDK[7]	DON[8]
	Means							
2015	129.8a	87.7a	6.8a	37.3a	80.3a	31.5a	6.9a	8.9b
2016	123.4b	83.7b	3.8b	22.5b	77.0b	17.3b	5.3b	13.2a
	ANOVA							
Genotype	28.9**	156.5**	4.4**	243.0**	364.5**	276.5**	24.3**	60.4**
Year	10554.1**	4207.6**	2355.9**	56177.3**	2899.2**	50892.7**	643.7**	4522.4**
G x Y	10.5**	33.1**	1.7**	166.8**	288.5**	196.8**	7.6**	14.4**
CV[9]	1.6	5.2	20.0	33.1	16.7	41.3	27.7	28.0
	Broad sense heritability							
h^2	0.64	0.79	0.60	0.31	0.20	0.28	0.69	0.77
LL	0.59	0.75	0.55	0.21	0.08	0.17	0.64	0.73
UL	0.71	0.83	0.69	0.45	0.35	0.42	0.75	0.81

** $p \leq 0.05$.
[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).
Within columns, means followed by the same letter are not significantly different according to a *t* test (0.05).
[9]CV, coefficient of variation

Table 3.2. Pearson correlations among traits evaluated in a 256 entry wheat mapping panel in 2015 and 2016, Lexington, KY. Correlations from 2015 are above the diagonal; 2016 correlations are below diagonal.

		2015							
	Traits	HD[1]	PH[2]	Rating[3]	SEV[4]	INC[5]	Index[6]	FDK[7]	DON[8]
2016	HD	.	0.26**	0.08ns	0.34**	0.20**	0.33**	0.06ns	0.13**
	PH	0.51**	.	-0.38**	-0.20**	-0.30**	-0.25**	-0.32**	-0.29**
	Rating	-0.22**	-0.35**	.	0.59**	0.73**	0.67**	0.50**	0.43**
	SEV	-0.30**	-0.34**	0.50**	.	0.56**	0.97**	0.52**	0.46**
	INC	0.14**	0.04ns	0.11*	0.03ns	.	0.72**	0.46**	0.42**
	Index	-0.17**	-0.26**	0.47**	0.86**	0.51**	.	0.56**	0.50**
	FDK	-0.29**	-0.43**	0.56**	0.50**	-0.01ns	0.42**	.	0.71**
	DON	0.11*	-0.13**	0.35**	0.36**	-0.02ns	0.29**	0.59**	.

** $p \leq 0.01$, * $p \leq 0.05$, ns nonsignificant.

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Table 3.3. GWAS of 250 soft red winter wheat lines grown in 2015-2016, Lexington, KY. Only SNPs with LOD score > 3 ($p<0.001$) are shown. Effect of SNP is expressed in percentage of the mean of each trait.

Trait	Year	SNP	Chr[1]	*p* value	Effect (%)	cM[2]	MAF[3]
PH[4]	Average	M10186	6A	0.00071	-2.14	190.27	0.112
	2015	.	.	.	.	.	.
	2016	M11214	6B	0.00064	-1.66	245.09	0.444
Rating[5]	Average	M12955*	7B	0.00045	0.39	359.99	0.13
		M12960*	7B	0.00098	0.35	379.94	0.142
		M12143	7A	0.00100	-0.33	398.23	0.152
	2015	.	.	.	.	.	.
	2016	M1458	1B	0.00031	-0.38	238.2	0.404
		M7852	5A	0.00055	0.38	79.39	0.268
		M1318	1B	0.00073	0.01	7.67	0.486
		M1461	1B	0.00087	-0.37	238.2	0.306
SEV[6]	Average	.	.	.	.	.	.
	2015	M9385*	5B	0.00010	-5.04	433.05	0.152
		M5248*	3A	0.00029	-3.93	284.21	0.282
		M2347	1D	0.00068	-3.39	435.16	0.412
		M1657	1B	0.00087	-4.84	269.73	0.13
		M654	1A	0.00087	-0.50	440.55	0.494
	2016	M6781	4A	0.00005	-2.51	147.89	0.124
		M11507	6B	0.00032	-1.56	410.92	0.342
		M12936	7B	0.00036	-1.58	321.13	0.294
		M11498	7B	0.00083	1.37	408.89	0.398
INC[7]	Average	M12955*	7B	0.00008	4.01	359.99	0.13
		M12960*	7B	0.00016	3.76	379.94	0.142
		M5559	3A	0.00046	2.86	433.76	0.23
		M12957*	7B	0.00056	3.10	362.32	0.172
		M5589	3A	0.00063	2.63	513.89	0.25
	2015	M3268*	2B	0.00020	-3.91	240.19	0.328
		M12960*	7B	0.00040	5.02	379.94	0.142
	2016	M7272	4B	0.00004	3.63	71.97	0.408
		M7271	4B	0.00032	3.35	63.55	0.318
		M2050	1D	0.00040	3.94	5.47	0.172
Index	Average	M12955*	7B	0.00040	3.05	359.99	0.13
		M12957*	7B	0.00066	2.60	362.32	0.172

[1]Chr., chromosome; [2]cM, Centimorgan; [3]MAF, minor allele frequency; [4]PH, plant height; [5]Rating, FHB Rating plant; [6]SEV, severity; [7]INC, incidence; [8]FDK, Fusarium damaged kernel; [9]DON, deoxynivalenol, *, markers with pleotropic effect.

Table 3.3. Continued

Trait	Year	SNP	Chr[1]	*p* value	Effect (%)	cM[2]	MAF[3]
Index	Average	M12960*	7B	0.00081	2.79	379.94	0.142
	2015	M9385*	5B	0.00021	-5.30	433.05	0.152
		M5248*	3A	0.00038	-4.26	284.21	0.282
		M3268*	2B	0.00045	-3.91	240.19	0.328
		M7759	4B	0.00083	3.56	327.27	0.462
	2016	.	.	.	.	.	.
FDK[8]	Average	M9432*	5B	0.00059	-0.64	447.74	0.304
		M11423	6B	0.00084	-0.86	375.21	0.144
	2015	M2926	2A	0.00017	-1.05	413.64	0.264
		M9385*	5B	0.00087	-1.09	433.05	0.152
	2016	M11046	6B	0.00056	0.63	226.64	0.388
		M2977	2A	0.00071	0.78	473.89	0.164
DON[9]	Average	M9432*	5B	0.00002	-1.42	447.74	0.304
		M6959	4A	0.00082	-1.17	356.78	0.264
	2015	.	.	.	.	.	.
	2016	M13271	7D	0.00011	2.53	337.29	0.112
		M9432*	5B	0.00031	-1.47	447.74	0.304

[1]Chr., chromosome; [2]cM, Centimorgan; [3]MAF, minor allele frequency; [4]PH, plant height; [5]Rating, FHB Rating plant; [6]SEV, severity; [7]INC, incidence; [8]FDK, Fusarium damaged kernel; [9]DON, deoxynivalenol, *, markers with pleotropic effect.

Table 3.4. Quantitative trait locus (QTL) contrasting alleles effects on plant height (PH), heading date (HD), FHB rating (Rating), Fusarium damaged kernels (FDK) and deoxynivalenol (DON) means of 250 soft red winter wheat lines grown in 2015 and 2016 in Lexington, KY.

QTL[1]	Number of lines	PH		HD		Rating		FDK		DON	
no Fhb1[2]	229	85.8	A	126.6	A	5.3	A	6.1	A	11.1	A
Fhb1[3]	19	84.6	A	126.4	A	5.4	A	6.1	A	10.5	A
Rht-B1a[4]	121	85.6	A	126.9	A	5.5	A	6.2	A	12.1	A
Rht-B1b[5]	125	85.8	A	126.3	A	5.1	B	5.9	A	10.1	B
Rht-D1a[6]	157	85.9	A	126.3	B	5.1	B	5.9	A	10.2	B
Rht-D1b[7]	88	85.1	A	127.1	A	5.6	A	6.5	A	12.9	A
Vrn-A1[8]	236	85.7	A	126.6	A	5.3	A	6.0	A	11.0	A
Vrn-A1-short[9]	14	84.7	A	127.0	A	5.2	A	7.0	A	11.9	A
*Vrn-B1**[10]	242	85.8	A	126.7	A	5.3	A	6.0	A	11.0	A
Vrn-B1-short[11]	5	82.6	B	125.1	B	5.5	A	5.7	A	8.6	B
Vrn-D3b[12]	171	85.5	A	126.7	A	5.4	A	6.3	A	11.6	A
Vrn-D3a-early[13]	76	86.1	A	126.5	A	4.9	B	5.6	A	9.9	B
Ppd-A1a[14]	140	85.6	A	126.1	B	5.2	A	5.9	A	10.4	B
Ppd-A1b[15]	104	85.9	A	127.4	A	5.4	A	6.2	A	11.9	A
Ppd-B1a[16]	19	85.1	A	126.3	A	5.6	A	6.4	A	11.2	A
Ppd-B1b[17]	188	85.9	A	126.7	A	5.2	A	6.0	A	11.1	A
Ppd-D1a[18]	123	84.4	B	126.2	B	5.5	A	6.4	A	11.8	A
Ppd-D1b[19]	123	87.2	A	127.0	A	5.0	B	5.7	B	10.3	B

[1]QTL, quantitative trait loci; [2]*Fhb1*-S, susceptible; [3]*Fhb1*-R, resistant; [4]*Rht-B1a* and [6]*Rht-D1a*, height wild type allele; [5]*Rht-B1b* and [7]*Rht-D1b*, dwarfing height allele; [8]*Vrn-A1*, [10]*Vrn-B1* and [12]*Vrn-D3*, wild type alleles; [9]V*rn-A1-short*, [11]*Vrn-B1-short* and [13]*Vrn-D3-early*, mutant allele; [14]*Ppd-A1a*, [16]*Ppd-B1a* and [18]*Ppd-D1a*, photoperiod insensitive; [15]*Ppd-A1b*, [17]*Ppd-B1b* and [19]*Ppd-D1b,* photoperiod sensitive.
Within columns, means followed by the same letter are not significantly different according to *t* test (0.05).
*Within column, means followed by the same letter are not significant different according to Welch's t test (0.05).

Table 3.5. Means for scab traits for contrasting alleles at SNPs M9432, M6959, and M11423, associated with Fusarium damaged kernels (FDK) and deoxynivalenol (DON) in 250 soft red winter wheat lines grown in 2015 and 2016, Lexington, KY. Number of lines for TT and AA genotypes at each SNP is shown in parenthesis.

Trait	M9432 (Chr. 5B)		M6959 (Chr. 4A)		M11423 (Chr. 6B)	
	TT (174)	AA (76)	TT (183)	AA (65)	TT (213)	AA (35)
FDK[1]	5.7b	6.9a	6.0a	6.3a	5.9b	7.2a
DON[2]	10.4b	12.5a	10.7b	12.2a	10.6b	13.8a
Rating[3]	5.2a	5.5a	5.3a	5.3a	5.2a	5.5a
SEV[4]	29.1a	31.2a	29.6a	30.3a	29.3b	32.8a
INC[5]	78.3a	79.3a	78.4a	79.1a	77.7b	83.2a
Index[6]	23.7a	25.8a	24.1a	25.0a	23.6b	28.0a

[1]FDK, Fusarium damaged kernel (%); [2]DON, deoxynivalenol (ppm); [3]FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%).
Within rows at each SNP, means followed by the same letter are not significantly different according to a *t* test ($p < 0.05$).

Table 3.6. Performance of 20 soft red winter wheat breeding lines and cultivars with the lowest deoxynivalenol (DON, ppm) concentration and their respective plant height (PH, cm), heading date (HD, Julian date), and genotypes at these QTL: *Rht-B1*, *Rht-D1*, *Vrn-A1*, *Vrn-B1*, *Vrn-D3*, *Ppd-A1*, *Ppd-B1*, *Ppd-D1* and *Fhb1*. Plant height and heading date are average values for 2015 and 2016, Lexington, KY.

Genotypes	DON	Rank	PH	HD	*Rht-B1*	*Rht-D1*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3*	*Ppd-A1*	*Ppd-B1*	*Ppd-D1*	*Fhb1*
IL06-13708	3.7	1	90.2	123.0	b[2]	a[1]	W[3]	W	*vrn-D3a-early*[5]	b	b[8]	b	S[9]
04620A1-1-7-4	4.0	2	85.7	124.5	b	a	W	W	*vrn-D3a-early*	b	a	b	S
KY02C-2215-02	4.5	3	83.2	126.8	a	a	W-short[4]	W	*vrn-D3*b	a[7]	b	a	S
MO080584	4.6	4	110.5	132.5	b	a	W	W	*vrn-D3a-early*	a	b	b	S
07287RA1-14	4.7	5	84.5	123.3	b	a	W	W	*vrn-D3a-early*	a	b	a	S
KY02C-1121-75	4.7	6	89.5	127.8	a	b	W	W	*vrn-D3*b[6]	a	b	b	S
IL08-34020	4.8	7	82.6	125.3	b	a	W	W	*vrn-D3a-early*	a	b	a	S
KY02C-1058-03	4.8	8	87.0	128.3	a	b	W	W	*vrn-D3*b	b	b	a	S
MO081699	5.0	9	93.4	124.3	a	a	W	W	*vrn-D3*b	a	b	a	S
05287A1-1-13	5.0	10	83.2	123.0	b	a	W	W	*vrn-D3*b	a	b	b	S
OH08-207-33	5.1	11	83.8	123.3	a	a	W	W	*vrn-D3a-early*	b	a	b	S
MO100745	5.1	12	100.3	133.3	b	a	W	W	*vrn-D3a-early*	a	b	b	S
IL06-7550	5.2	13	90.2	126.8	b	a	W	W	*vrn-D3a-early*	b	b	b	S
04719A1-16-1-1-7	5.2	14	79.4	124.5	b	a	W	W	*vrn-D3a-early*	a	b	a	S
KY05C-1381-77-7-5	5.3	15	87.6	124.8	b	a	W	W	*vrn-D3*b	b	b	a	S
MO090574	5.4	16	95.3	124.8	a	a	W	W	*vrn-D3*b	a	b	a	S
OH08-234-4	5.5	17	90.2	125.8	a	a	W	W	*vrn-D3a-early*	a	--	b	S
KY06C-1003-139-8-3	5.6	18	80.0	124.8	b	a	W	W	*vrn-D3*b	b	b	a	S
IL01-11934	5.7	19	76.2	124.0	b	a	W	W	*vrn-D3a-early*	a	b	b	S
KY03C-2047-06	5.7	20	80.7	124.0	a	b	W	W	*vrn-D3*b	a	b	a	S
Average	5.0		87.7	125.7									

[1]a, wild allele for Rht-B1 and Rht-D1; [2]b, semi dwarf allele for Rht-B1 and Rht-D1; [3]w, winter allele for *Vrn-A1, Vrn-B1*, [4]w-short, short vernalization requirement; [5]*vrn-D3a-early*, early flowering, [6]*vrn-D3b*, wild type allele; [7]a, photoperiod insensitive allele for *Ppd-A1, Ppd-B1,* and *Ppd-D1*; [8]b, photoperiod sensitive allele for *Ppd-A1, Ppd-B1, and Ppd-D1*, [9]S, susceptible allele for *FHB1*; --, no genotypic data.

Table 3.7. Performance of 20 soft red winter wheat breeding lines and cultivars with the highest deoxynivalenol (DON, ppm) concentration and their respective plant height (PH, cm), heading date (HD, Julian date), and genotypes at these QTL: *Rht-B1*, *Rht-D1*, *Vrn-A1*, *Vrn-B1*, *Vrn-D3*, *Ppd-A1*, *Ppd-B1*, *Ppd-D1*, and *Fhb1*. Plant height and heading date are average values for 2015 and 2016, Lexington, KY.

Genotype	DON	Rank	PH	HD	*Rht-B1*	*Rht-D1*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3*	*Ppd-A1*	*Ppd-B1*	*Ppd-D1*	Fhb1
REDRUBY	18.2	241	80.7	127.5	a[1]	b[2]	W[3]	W	*vrn-D3*b	b	b[8]	a	S[9]
OH08-149-11	18.3	242	87.7	127.3	b	a	W	W	*vrn-D3a-early*[5]	b	a	b	S
MD03W61-11-2	18.5	243	80.7	124.3	a	b	W-short[4]	W	*vrn-D3*b[6]	a[7]	--	a	S
D6234	18.6	244	90.2	130.5	a	b	W	W	*vrn-D3*b	b	b	a	S
VA10W-119	18.9	244	79.4	128.5	a	b	W	W	*vrn-D3*b	b	b	a	S
MD03W485-10-2	19.1	245	79.4	128.5	a	b	W	W	*vrn-D3*b	b	--	a	S
SS5205	19.4	246	74.9	128.8	a	b	W	W	*vrn-D3a-early*	b	a	a	S
VA10W-140	19.6	247	78.7	124.3	a	b	W	W	*vrn-D3*b	a	b	a	S
VA10W-21	19.7	248	76.8	126.8	a	b	W	W	*vrn-D3*b	b	b	a	S
OH08-133-25	19.8	249	91.4	127.3	a	a	W	W	*vrn-D3*b	a	a	b	S
VA06W-412	19.8	250	74.9	124.0	a	b	W	W	*vrn-D3*b	a	b	b	S
IL07-12948	20.1	251	68.0	126.3	b	a	W	W	*vrn-D3a-early*	b	a	b	S
MD03W665-10-3	20.9	252	86.4	128.8	a	b	W	W	*vrn-D3*b	b	--	a	S
E6012	22.0	254	88.3	128.3	a	b	W	W	*vrn-D3*b	b	b	a	S
E2041	22.3	255	84.5	127.5	a	b	W	W	*vrn-D3*b	b	b	a	S
VA08W-613	23.7	256	81.3	126.8	a	b	W-short	W	*vrn-D3a-early*	b	b	a	S
SSMPV57	23.8	257	82.6	123.0	a	a	W	W	*vrn-D3*b	a	b	a	S
VA10W-125	26.4	258	82.6	123.8	a	b	W	W-short	*vrn-D3*b	b	b	a	S
D8006	26.8	259	85.1	128.8	a	b	W	W	*vrn-D3*b	b	b	a	S
CRYSTAL	33.3	260	76.2	128.8	a	b	W	W	*vrn-D3*b	b	b	a	S
Average	21.6		81.5	126.9									

[1]a, wild allele for Rht-B1 and Rht-D1; [2]b, semi dwarf allele for Rht-B1 and Rht-D1; [3]w, winter allele for *Vrn-A1, Vrn-B1*, [4]w-short, short vernalization requirement; [5]*vrn-D3a-early*, early flowering, [6]*vrn-D3b*, wild type allele; [7]a, photoperiod insensitive allele for *Ppd-A1, Ppd-B1,* and *Ppd-D1*; [8]b, photoperiod sensitive allele for *Ppd-A1, Ppd-B1, and Ppd-D1*, [9]S, susceptible allele for *FHB1*; --, no genotypic data.

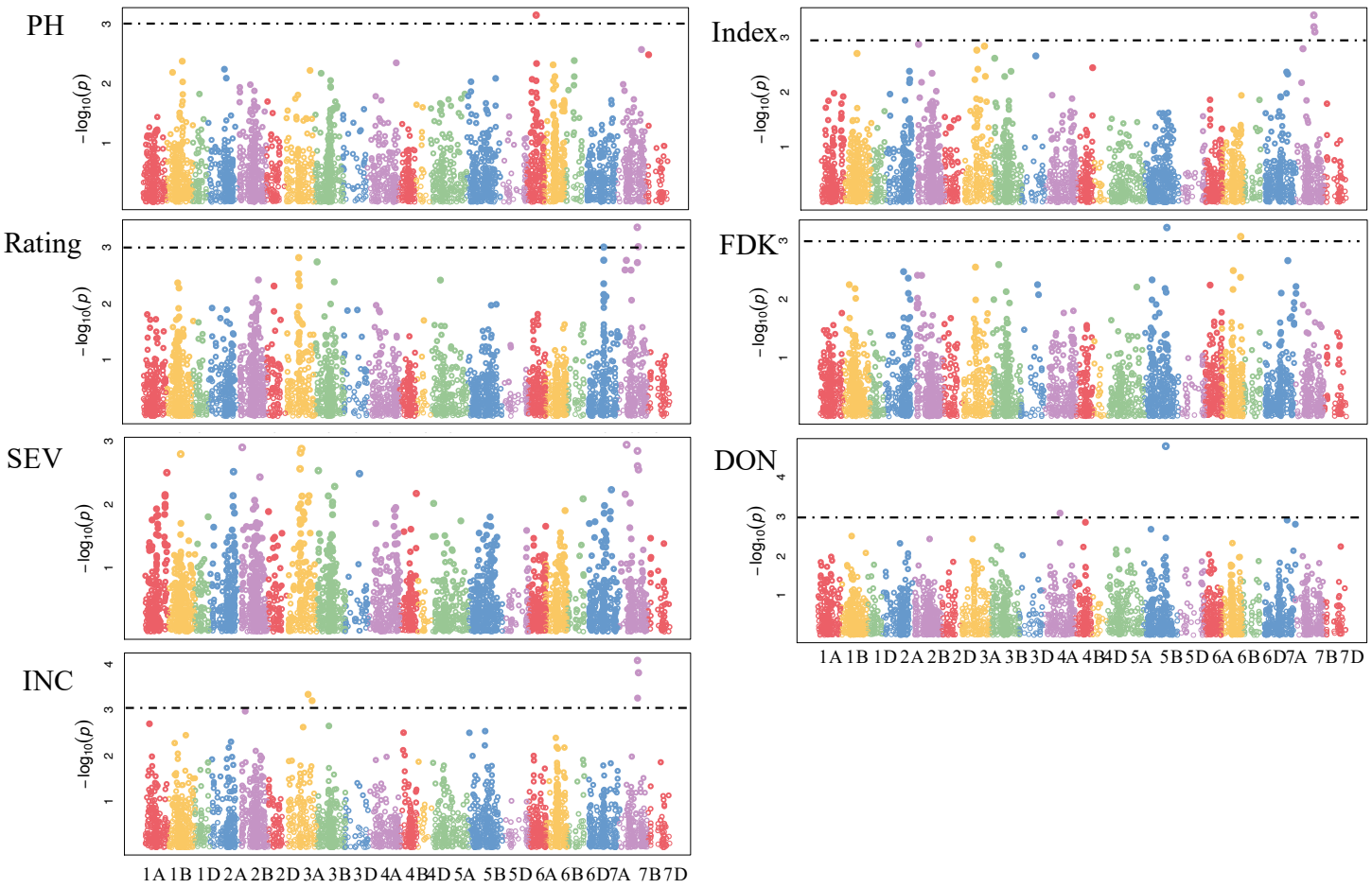


Figure 3.1. Manhattan plots of genome-wide association study (GWAS) for plant height (PH), FHB rating, severity (SEV), incidence (INC), Index, Fusarium damaged kernels (FDK), and deoxynivalenol (DON) in 250 soft red winter wheat cultivars and breeding lines from the T-CAP panel grown in 2015-2016, Lexington, KY.

Supplemental Table S3.1. Means for heading date (HD), plant height (PH), rating (RAT), severity (SEV), incidence (INC), index, Fusarium damaged kernels (FDK) and deoxynivalenol (DON) for each line evaluate in 2015-2016, Lexington, KY.

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
INDIANA	011007A1-14-16-50	124.75	74.30	6.50	27.73	62.50	18.71	7.24	8.78
INDIANA	0175A1-37-4-1	127.25	83.82	6.25	38.29	88.75	34.57	9.83	12.78
INDIANA	02444A1-23-1-3	128.75	83.82	5.00	29.51	82.50	25.50	7.09	10.05
INDIANA	03207A1-7-3-1	124.00	78.74	7.25	28.62	78.75	23.60	5.87	10.78
INDIANA	03549A1-18-25	124.50	83.19	5.25	34.45	67.50	26.09	6.22	11.88
INDIANA	03633A1-69-2-5	124.00	71.76	6.50	39.23	87.50	34.15	7.61	12.88
INDIANA	04606RA1-1-7-1	124.25	85.09	5.00	27.56	96.25	26.18	7.99	7.03
INDIANA	04606RA1-1-7-1-6	124.75	93.98	4.50	20.01	57.50	11.00	5.35	10.50
INDIANA	04620A1-1-7-4	124.50	85.73	4.25	20.18	72.50	15.01	2.27	3.98
INDIANA	04702A1-18	125.00	83.82	5.25	27.23	82.50	22.26	9.86	12.00
INDIANA	04719A1-16-1-1-7	124.50	79.38	5.00	23.44	66.25	16.05	2.40	5.20
INDIANA	0513A1-1-3	123.25	80.01	6.00	23.94	76.25	18.56	6.00	8.15
INDIANA	05222A1-1-2-1	124.75	87.63	4.75	23.11	77.50	17.81	5.68	10.20
INDIANA	05247A1-7-3-120	126.00	79.38	6.25	30.73	92.50	27.60	7.16	10.75
INDIANA	05247A1-7-3-27	122.75	80.01	6.75	38.28	87.50	34.75	7.00	7.68
INDIANA	05247A1-7-7-3-1	123.75	85.09	4.50	27.05	81.25	21.89	5.02	6.95
INDIANA	05251A1-1-136-9-5	120.25	68.58	7.75	60.04	92.50	55.66	9.81	7.95
INDIANA	05264A1-1-3-2	125.50	83.82	5.50	22.38	87.50	19.71	8.62	10.05
INDIANA	05287A1-1-13	123.00	83.19	4.00	18.02	63.75	11.30	4.54	5.00
INDIANA	0537A1-12	124.75	83.82	5.50	38.69	75.00	27.96	7.04	8.90
INDIANA	0537A1-3-12	126.50	81.92	3.75	27.47	65.00	16.53	2.74	6.50
INDIANA	0566A1-3-1-65	128.75	90.17	4.50	21.68	77.50	18.48	5.36	9.88
INDIANA	0566A1-3-1-67	128.25	79.38	6.25	43.96	77.50	36.58	7.14	8.28

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
INDIANA	0570A1-2-39-5	125.50	81.28	5.75	29.51	68.75	21.67	6.74	9.75
INDIANA	06403A1-4	124.50	88.27	6.00	31.56	73.75	22.58	10.34	11.78
INDIANA	0722A1-1-7	124.00	86.36	5.75	31.88	88.75	28.44	8.37	14.88
INDIANA	07287RA1-14	123.25	84.46	4.50	23.36	83.75	18.92	6.03	4.68
INDIANA	07290A1-12	128.75	82.55	6.00	37.73	85.00	33.30	6.89	13.33
INDIANA	0762A1-2-8	124.75	81.92	5.75	29.11	82.50	24.43	5.09	10.90
CIMMYT	91193	125.25	83.82	5.50	26.19	78.75	20.49	9.71	14.75
CIMMYT	92201	123.75	83.82	5.50	27.20	81.25	22.24	8.85	10.20
INDIANA	9346A1-2-5-5-2-1	124.00	85.73	4.75	23.10	81.25	19.25	4.63	8.70
OHIO	BECKER	125.50	76.84	7.00	52.38	90.00	48.64	9.16	17.58
MISSOURI	BESS	126.75	88.27	3.50	23.07	63.75	15.05	4.00	8.80
OHIO	BROMFIELD	129.00	90.17	3.00	16.78	70.00	11.84	2.88	6.33
NEW_YORK	CALEDONIA	133.00	88.90	5.50	42.22	91.25	38.85	8.90	18.10
MARYLAND	CATOCIN	131.75	89.54	4.00	26.80	70.00	17.76	3.11	7.78
NEW_YORK	CAYUGA	135.50	116.21	4.75	32.50	70.00	23.88	2.95	11.28
MARYLAND	CHOPTANK	124.00	83.82	5.50	36.66	73.75	29.23	6.85	12.50
INDIANA	CLARK	123.75	80.65	4.50	26.16	76.25	20.62	5.75	6.70
MICHIGAN	CRYSTAL	128.75	76.20	7.00	28.34	83.75	24.82	10.24	33.30
MICHIGAN	D6234	130.50	90.17	6.00	35.94	87.50	32.48	5.56	18.55
MICHIGAN	D8006	128.00	86.36	5.50	31.51	67.50	21.79	6.83	26.85
MICHIGAN	E2041	128.50	83.82	6.00	29.41	75.00	22.34	6.26	22.45
MICHIGAN	E5011	134.25	80.01	5.25	33.21	91.25	30.41	9.54	17.83
MICHIGAN	E5024	130.25	85.73	5.00	27.10	73.75	20.32	6.32	18.08
MONTANA	E6012	128.50	81.28	5.50	26.53	72.50	19.12	5.99	21.05

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
MISSOURI	ERNIE	123.50	85.09	5.50	25.44	55.00	14.33	7.56	11.88
KENTUCKY	FOSTER	126.50	87.00	6.00	33.98	87.50	30.67	7.09	7.33
NEW YORK	HOPKINS	133.00	93.98	4.00	30.54	67.50	20.65	5.37	13.73
ILLINOIS	IL00-8109	125.00	87.63	4.50	31.93	75.00	24.28	4.79	12.05
ILLINOIS	IL00-8530	123.00	78.74	5.00	26.57	66.25	17.12	4.30	6.68
ILLINOIS	IL00-8633	126.75	76.20	5.25	33.45	91.25	30.70	8.18	14.43
ILLINOIS	IL01-11934	124.00	76.20	6.00	23.46	85.00	19.88	4.78	5.73
ILLINOIS	IL02-18228	126.75	81.92	5.50	42.18	88.75	37.79	6.75	12.93
ILLINOIS	IL02-19483B	121.25	87.63	5.50	29.82	60.00	17.83	3.50	12.85
ILLINOIS	IL04-24668	124.00	82.55	5.25	30.34	76.25	23.81	6.53	10.98
ILLINOIS	IL04-9942	125.75	76.84	6.50	31.19	81.25	26.04	8.59	12.93
ILLINOIS	IL05-4236	127.25	83.19	5.75	26.98	82.50	23.04	6.39	13.98
ILLINOIS	IL06-13072	125.75	82.55	4.75	23.86	86.25	21.22	4.77	5.85
ILLINOIS	IL06-13708	123.00	90.17	2.75	23.50	83.75	19.95	4.34	3.68
ILLINOIS	IL06-13721	127.00	95.89	4.75	32.17	85.00	27.97	6.04	11.30
ILLINOIS	IL06-14262	127.50	98.43	4.00	21.63	57.50	12.98	3.78	7.55
ILLINOIS	IL06-14325	126.00	90.81	4.00	19.69	76.25	14.90	2.95	6.25
ILLINOIS	IL06-23571	128.75	100.33	4.75	29.59	86.25	26.09	6.13	11.23
ILLINOIS	IL06-31053	121.50	81.92	6.50	40.09	80.00	32.83	5.95	10.65
ILLINOIS	IL06-7550	126.75	90.17	3.75	27.05	62.50	16.75	2.56	5.18
ILLINOIS	IL06-7653	124.50	78.74	5.75	36.44	76.25	29.90	7.27	10.33
ILLINOIS	IL07-12948	126.25	67.95	6.50	58.30	93.75	55.66	11.05	20.13
ILLINOIS	IL07-16075	128.00	86.36	5.25	32.17	86.25	28.11	4.57	9.75
ILLINOIS	IL07-19334	127.50	85.09	3.00	24.30	76.25	19.49	4.42	6.83

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
ILLINOIS	IL07-20728	125.75	87.00	4.75	26.24	62.50	17.03	5.62	8.33
ILLINOIS	IL07-20743	126.50	86.36	5.00	18.45	77.50	14.68	4.83	6.85
ILLINOIS	IL07-21847	125.50	87.63	3.25	23.71	75.00	18.81	3.42	7.55
ILLINOIS	IL07-23420	126.50	83.82	6.00	30.32	80.00	24.40	6.64	10.40
ILLINOIS	IL07-24841	122.25	88.90	4.50	21.14	72.50	15.06	3.46	8.30
ILLINOIS	IL07-4415	129.75	98.43	3.75	27.92	75.00	21.13	3.09	6.78
ILLINOIS	IL07-6861	126.25	87.63	4.00	16.86	62.50	10.94	5.27	10.90
ILLINOIS	IL08-12174	124.00	75.57	6.50	34.50	72.50	25.31	11.56	17.45
ILLINOIS	IL08-12206	127.25	93.98	3.50	25.65	60.00	17.40	4.58	8.78
ILLINOIS	IL08-22075	124.00	90.81	4.75	27.44	85.00	23.41	5.37	10.13
ILLINOIS	IL08-31639	127.00	85.73	5.25	30.03	82.50	24.38	5.42	9.38
ILLINOIS	IL08-33373	130.50	92.08	3.25	18.13	78.75	14.01	3.73	9.00
ILLINOIS	IL08-33951	124.25	77.47	6.25	33.22	83.75	30.08	5.93	10.65
ILLINOIS	IL08-34020	125.25	82.55	5.00	15.58	75.00	11.10	2.76	4.75
ILLINOIS	IL08-9266	123.25	82.55	5.00	24.59	76.25	19.18	5.39	11.90
ILLINOIS	IL99-26442	126.25	83.82	5.25	24.02	90.00	22.06	5.53	8.40
INDIANA	INW0411	123.75	84.46	5.25	28.57	70.00	20.22	7.15	8.88
INDIANA	INW0412	129.75	90.81	4.00	28.06	82.50	23.87	3.78	12.33
INDIANA	INW1021	124.50	81.92	4.50	29.01	73.75	22.79	5.84	10.38
ARKANSAS	JAYPEE	130.75	98.43	5.25	25.11	82.50	21.06	3.57	12.95
KENTUCKY	KY02C-1058-03	128.25	87.00	3.75	22.04	90.00	19.65	3.18	4.83
KENTUCKY	KY02C-1076-07	128.25	83.82	6.25	31.99	81.25	25.64	4.64	10.50
KENTUCKY	KY02C-1121-75	127.75	89.54	3.75	19.03	75.00	15.07	2.41	4.73
KENTUCKY	KY02C-1122-06	126.75	89.54	5.50	31.31	87.50	27.44	7.72	7.50

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
KENTUCKY	KY02C-2215-02	126.75	83.19	3.75	18.66	77.50	14.10	2.75	4.50
KENTUCKY	KY03C-1002-02	123.75	81.92	3.50	18.51	63.75	11.73	4.56	6.65
KENTUCKY	KY03C-1192-37	128.25	92.08	4.75	25.36	81.25	20.07	4.39	8.93
KENTUCKY	KY03C-1195-10-1-5	124.25	78.74	5.25	14.33	77.50	11.07	5.60	9.58
KENTUCKY	KY03C-1221-01	124.75	83.19	5.25	22.99	80.00	18.14	5.37	9.13
KENTUCKY	KY03C-1221-06	129.75	91.44	4.75	26.21	71.25	17.21	3.16	8.40
KENTUCKY	KY03C-1221-22	126.75	81.92	5.25	17.60	85.00	14.68	3.04	8.43
KENTUCKY	KY03C-1237-01	124.25	81.28	6.50	31.93	71.25	25.93	7.18	12.88
KENTUCKY	KY03C-1237-32	128.00	85.09	5.25	26.01	87.50	23.18	3.24	6.13
KENTUCKY	KY03C-2047-02	125.00	76.84	6.75	35.38	76.25	29.34	5.35	8.35
KENTUCKY	KY03C-2047-06	124.00	80.65	6.00	23.93	77.50	18.87	4.21	5.73
KENTUCKY	KY03C-2049-02	124.25	85.73	6.50	29.29	81.25	23.82	5.68	8.88
KENTUCKY	KY03C-2314-08	126.50	85.73	4.50	20.13	81.25	16.69	4.26	7.90
KENTUCKY	KY03C-2399-02	125.00	80.01	3.75	23.36	66.25	15.84	9.08	9.03
KENTUCKY	KY04C-1128-38-1-5	126.00	88.90	6.00	22.66	81.25	18.97	4.36	10.20
KENTUCKY	KY04C-2006-41-1-1	124.00	78.11	5.50	30.30	78.75	24.05	5.42	14.55
KENTUCKY	KY04C-2151-40	127.75	80.01	5.50	27.25	86.25	24.15	6.64	6.65
KENTUCKY	KY04C-2151-41	125.75	83.82	6.00	30.87	77.50	23.79	5.11	13.38
KENTUCKY	KY04C-3006-33-14-3	130.00	87.00	5.25	30.43	96.25	29.07	4.70	7.85
KENTUCKY	KY05C-1007-2-12-5	127.00	75.57	6.50	34.65	91.25	31.37	4.54	9.08
KENTUCKY	KY05C-1105-42-20-1	123.25	73.66	7.75	29.45	86.25	25.40	7.86	10.80
KENTUCKY	KY05C-1381-77-7-5	124.75	87.63	4.50	20.99	92.50	19.33	2.97	5.28
KENTUCKY	KY05C-1617-17-17-3	127.75	85.73	6.50	39.44	88.75	35.12	9.29	14.43
KENTUCKY	KY06C-1003-139-8-3	124.75	80.01	5.75	20.67	72.50	14.86	4.56	5.55

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
KENTUCKY	KY93C-1238-17-1	124.50	89.54	6.25	28.52	85.00	24.90	8.47	10.78
OHIO	MALABAR	132.25	98.43	5.00	25.18	88.75	23.19	4.84	10.25
MARYLAND	MD01W270-10-3	124.75	78.74	5.75	31.56	68.75	23.55	4.79	11.25
MARYLAND	MD03W104-10-2	130.75	92.71	5.00	29.54	86.25	24.05	4.38	9.45
MARYLAND	MD03W151-10-12	130.50	100.33	5.25	33.57	82.50	27.09	3.81	9.78
MARYLAND	MD03W485-10-10	127.75	88.90	5.75	32.27	86.25	28.48	5.01	9.20
MARYLAND	MD03W485-10-12	128.25	85.73	5.25	36.49	83.75	32.42	4.55	6.80
MARYLAND	MD03W485-10-2	128.75	86.36	7.25	53.07	83.75	46.13	7.36	19.10
MARYLAND	MD03W485-10-8	130.75	100.97	4.50	32.50	75.00	24.21	4.70	8.08
MARYLAND	MD03W61-11-2	124.25	80.65	5.75	28.09	86.25	24.82	11.88	18.50
MARYLAND	MD03W61-11-3	128.75	90.81	5.50	33.78	86.25	29.40	3.82	11.10
MARYLAND	MD03W64-10-3	131.00	99.06	3.75	22.78	63.75	15.05	3.18	9.18
MARYLAND	MD03W665-10-3	124.75	76.84	7.75	43.42	75.00	35.79	12.83	20.88
MARYLAND	MD03W665-10-5	125.00	77.47	4.75	34.36	73.75	26.68	5.54	11.55
MARYLAND	MD04W1197-11-13	122.25	78.11	7.25	38.26	83.75	32.69	8.90	16.05
MARYLAND	MD04W249-11-12	130.50	99.06	5.50	30.36	78.75	24.45	5.84	9.65
MARYLAND	MD04W249-11-13	129.00	99.70	3.75	24.99	63.75	16.40	2.46	6.83
MARYLAND	MD04W249-11-5	123.50	83.19	6.25	27.10	75.00	19.68	4.26	9.33
MARYLAND	MD04W249-11-7	117.50	82.55	7.50	30.27	82.50	24.68	5.30	12.45
MARYLAND	MD04W359-11-10	126.50	82.55	6.25	37.07	86.25	32.28	6.28	12.48
MARYLAND	MD04W8-11-4	124.25	91.44	5.00	25.27	70.00	17.90	6.20	8.73
MARYLAND	MD05W10208-11-13	124.25	85.09	5.50	30.34	82.50	24.66	6.26	10.53
MARYLAND	MD05W10208-11-14	130.00	87.63	5.75	38.13	81.25	31.45	5.83	12.90
MARYLAND	MD05W10208-11-3	129.00	79.38	6.25	38.09	81.25	33.24	7.49	13.90

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
MARYLAND	MD05W10208-11-6	125.50	83.82	4.00	25.91	73.75	17.34	3.57	10.45
MARYLAND	MD05W10208-11-7	127.00	74.30	7.00	50.69	90.00	46.44	9.73	12.63
MARYLAND	MD05W10208-11-8	126.00	82.55	7.50	43.54	85.00	39.54	7.52	9.20
MARYLAND	MD05W1292-11-1	124.25	81.28	4.50	22.35	86.25	19.38	3.59	8.93
MARYLAND	MD05W1292-11-4	133.00	100.33	3.50	18.71	66.25	12.35	1.93	7.13
MARYLAND	MD05W1317-11-4	126.25	87.63	6.25	36.67	86.25	33.19	5.77	10.23
MARYLAND	MD05W479-B-11-3	127.00	85.09	4.75	24.80	86.25	21.17	5.57	10.30
MARYLAND	MD07W272-11-5	125.25	76.84	7.00	25.43	83.75	21.97	5.82	9.60
MARYLAND	MD07W419UM5-11-11	125.75	81.92	6.00	42.32	76.25	33.48	11.93	14.78
MARYLAND	MD07W419UM5-11-12	125.50	80.65	5.75	33.38	93.75	31.40	6.31	12.63
MARYLAND	MD665-09-6	123.75	76.84	6.25	38.35	80.00	33.00	4.00	8.83
NEW YORK	MEDINA	134.75	98.43	6.25	48.29	83.75	43.34	6.77	12.48
VIRGINIA	MERL	125.00	80.65	5.75	24.49	77.50	18.83	7.24	15.95
MISSOURI	MO050921	135.25	91.44	3.50	30.94	77.50	23.86	3.51	8.80
MISSOURI	MO080103	124.00	88.90	3.50	22.51	55.00	12.57	4.01	6.80
MISSOURI	MO080104	125.50	89.54	3.25	17.91	68.75	12.44	3.49	5.98
MISSOURI	MO080584	132.50	110.49	2.00	14.10	58.75	8.35	1.88	4.63
MISSOURI	MO080589	128.25	90.81	4.50	17.51	71.25	12.20	4.09	10.80
MISSOURI	MO080864	131.25	94.62	4.25	20.17	78.75	15.83	4.07	12.28
MISSOURI	MO081163	128.75	83.82	7.00	37.50	90.00	34.11	4.78	10.40
MISSOURI	MO081280	128.75	88.27	5.75	38.57	91.25	35.63	8.87	11.08
MISSOURI	MO081559	131.25	91.44	3.25	23.01	70.00	17.05	3.85	7.93
MISSOURI	MO081652	124.50	85.09	4.50	24.25	73.75	17.60	4.41	6.05
MISSOURI	MO081699	124.25	93.35	2.75	16.19	52.50	8.17	3.01	4.98

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
MISSOURI	MO090574	124.75	95.25	3.25	20.10	66.25	12.77	2.87	5.40
MISSOURI	MO090581	124.50	96.52	4.00	17.71	67.50	12.06	3.94	10.40
MISSOURI	MO090821	127.00	92.71	2.75	13.51	45.00	5.95	2.76	5.93
MISSOURI	MO091011	129.25	90.17	3.75	27.40	68.75	18.39	2.95	10.90
MISSOURI	MO091159	124.75	87.00	4.75	18.19	52.50	9.68	4.53	7.20
MISSOURI	MO100172	123.75	87.00	5.50	22.30	70.00	16.16	3.75	9.60
MISSOURI	MO100231	124.25	76.20	7.50	61.26	88.75	56.99	15.30	15.63
MISSOURI	MO100265	124.50	93.35	4.50	22.86	63.75	14.89	2.99	7.38
MISSOURI	MO100519	127.00	85.09	6.50	32.61	87.50	28.86	6.98	17.60
MISSOURI	MO100535	124.25	82.55	6.75	34.78	92.50	32.44	6.77	11.88
MISSOURI	MO100539	128.25	86.36	5.75	34.69	87.50	31.00	6.87	10.30
MISSOURI	MO100647	127.00	83.82	5.50	34.42	78.75	29.30	7.64	12.90
MISSOURI	MO100745	133.25	100.33	2.50	17.42	73.75	12.72	1.79	5.10
MISSOURI	MO101329	129.25	81.28	6.50	35.18	90.00	32.54	6.18	14.00
MISSOURI	MO101358	126.00	78.11	7.00	42.79	80.00	35.17	14.10	14.30
MISSOURI	MO101361	124.00	81.92	6.00	24.38	83.75	20.58	4.78	8.25
NEW YORK	NY103-208-7263	137.00	100.97	6.00	47.79	78.75	40.45	5.10	10.10
NEW YORK	NY91017-8080	131.00	91.44	6.00	30.01	82.50	25.43	5.66	11.80
NEW YORK	NY96009-3037	135.50	100.97	5.25	35.43	81.25	29.74	4.70	13.68
NEW YORK	NY99066-3444	136.50	100.97	5.00	38.18	80.00	33.15	5.70	12.10
OHIO	OH05-200-74	130.50	90.17	5.00	30.26	82.50	25.31	6.63	13.90
OHIO	OH06-150-57	129.00	83.82	6.00	35.40	75.00	26.29	7.32	13.60
OHIO	OH06-180-57	128.00	95.25	5.50	30.52	85.00	25.72	4.74	13.20
OHIO	OH07-166-41	129.25	91.44	4.75	25.33	80.00	20.61	5.21	12.43

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
OHIO	OH07-166-49	128.75	87.63	5.75	30.00	75.00	22.70	8.20	16.18
OHIO	OH07-174-11	127.00	83.19	4.75	23.80	71.25	16.67	7.78	9.90
OHIO	OH07-238-15	130.75	93.98	5.25	33.45	86.25	27.91	7.33	16.10
OHIO	OH07-254-11	129.25	76.84	6.50	39.84	86.25	33.81	7.75	16.55
OHIO	OH07-263-3	124.25	90.81	4.00	29.24	57.50	16.66	4.31	9.13
OHIO	OH07-94-70	127.50	86.36	3.75	24.88	80.00	18.70	4.63	8.50
OHIO	OH07-95-7	127.00	89.54	4.75	27.36	88.75	23.80	4.82	11.13
OHIO	OH07-98-21	129.25	96.52	6.25	39.30	86.25	34.02	7.42	11.25
OHIO	OH08-101-57	129.75	93.35	4.25	18.46	65.00	12.58	2.48	7.28
OHIO	OH08-101-72	128.25	93.98	3.25	19.78	51.25	9.87	2.75	7.35
OHIO	OH08-107-16	134.00	83.19	4.25	21.12	82.50	18.02	3.47	8.50
OHIO	OH08-133-25	127.25	91.44	6.50	30.73	90.00	28.28	9.40	19.78
OHIO	OH08-141-6	122.75	89.54	5.00	29.54	77.50	22.55	5.25	7.20
OHIO	OH08-149-11	127.25	87.63	4.75	27.86	88.75	24.95	8.15	18.33
OHIO	OH08-161-4	124.00	81.28	5.75	27.03	85.00	22.63	6.99	7.73
OHIO	OH08-161-78	122.75	80.65	5.50	39.67	80.00	32.10	7.46	7.65
OHIO	OH08-170-66	128.75	89.54	5.50	35.96	91.25	33.17	7.17	15.20
OHIO	OH08-172-42	127.25	90.81	5.50	29.34	86.25	25.45	7.18	15.33
OHIO	OH08-178-52	124.00	89.54	4.75	22.46	82.50	18.47	3.83	8.75
OHIO	OH08-180-48	130.25	81.92	5.50	29.54	78.75	24.17	6.19	12.73
OHIO	OH08-182-4	125.25	83.82	5.00	23.90	72.50	17.53	4.40	11.20
OHIO	OH08-199-1	124.75	85.09	4.00	22.26	65.00	14.39	4.11	7.83
OHIO	OH08-206-19	127.25	93.35	5.00	21.62	88.75	19.52	4.93	9.40
OHIO	OH08-207-33	123.25	83.82	3.75	23.58	76.25	17.89	3.22	5.10

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
OHIO	OH08-234-4	125.75	90.17	4.00	21.88	61.25	15.76	4.54	5.53
OHIO	OH08-235-33	129.75	92.08	5.75	37.48	76.25	34.41	6.13	11.40
OHIO	OH08-246-15	126.75	81.92	5.50	32.48	90.00	29.33	5.30	12.05
OHIO	OH08-254-22	132.75	94.62	4.75	22.58	68.75	15.66	4.23	10.70
OHIO	OH08-256-47	130.75	94.62	6.75	38.98	95.00	37.85	13.11	17.00
OHIO	OH08-265-37	124.00	88.27	5.25	28.25	76.25	21.83	7.71	11.83
OHIO	OH08-269-58	128.50	92.71	5.75	40.82	83.75	35.24	10.54	12.00
OHIO	OH08-98-13	133.00	92.71	2.50	12.89	67.50	7.55	1.84	7.25
KENTUCKY	PEMBROKE	125.00	82.55	7.50	40.20	92.50	37.75	6.91	7.58
-	PIO 25R26	124.50	82.55	6.25	25.27	81.25	20.46	8.17	9.70
MICHIGAN	REDRUBY	127.50	80.65	6.25	32.71	92.50	30.77	10.22	18.18
VIRGINIA	SHIRLEY	122.25	78.11	6.25	29.72	77.50	24.02	5.70	10.38
VIRGINIA	SS520	126.00	75.57	5.50	38.24	82.50	33.88	7.21	13.88
VIRGINIA	SS5205	128.75	74.93	5.50	34.20	82.50	29.84	8.06	19.35
VIRGINIA	SSMPV57	123.00	82.55	6.50	31.46	71.25	23.78	9.91	23.80
MISSOURI	TRUMAN	134.75	92.71	3.50	20.95	58.75	12.16	2.77	6.33
VIRGINIA	USG3315	124.50	85.73	4.75	25.26	88.75	22.01	4.40	9.45
VIRGINIA	VA05W-151	124.50	77.47	6.00	34.62	88.75	32.15	3.06	8.93
VIRGINIA	VA05W-251	125.00	80.65	6.50	41.84	81.25	34.89	14.74	16.98
VIRGINIA	VA06W-412	124.00	74.93	6.75	38.82	88.75	34.54	10.48	19.78
VIRGINIA	VA07W-415	123.50	90.17	4.50	21.60	61.25	13.17	4.53	11.68
VIRGINIA	VA08MAS-369	125.25	81.28	5.00	33.84	81.25	28.13	4.31	8.70
VIRGINIA	VA08W-176	123.00	75.57	5.00	38.93	70.00	29.52	10.15	16.03
VIRGINIA	VA08W-294	123.75	80.65	4.75	27.23	86.25	23.37	4.94	9.43

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.1. Continued

STATE	NAME	HD[1]	PH[2]	RAT[3]	SEV[4]	INC[5]	IND[6]	FDK[7]	DON[8]
VIRGINIA	VA08W-613	126.75	81.28	5.50	42.22	83.75	37.82	13.06	23.70
VIRGINIA	VA09W-110	125.00	79.38	5.25	32.00	86.25	28.42	8.84	12.60
VIRGINIA	VA09W-112	123.25	80.65	5.75	35.60	71.25	26.14	6.76	9.70
VIRGINIA	VA09W-114	122.25	81.28	7.50	30.79	77.50	23.90	9.45	18.03
VIRGINIA	VA09W-188WS	126.00	73.03	6.00	40.26	78.75	35.58	8.90	16.35
VIRGINIA	VA09W-46	123.75	83.82	5.75	30.17	73.75	22.29	6.44	10.78
VIRGINIA	VA09W-52	126.25	83.82	6.00	45.31	80.00	40.52	8.74	13.80
VIRGINIA	VA09W-69	126.75	79.38	6.25	33.63	90.00	31.07	7.89	15.18
VIRGINIA	VA09W-73	123.00	81.28	5.50	25.15	72.50	18.51	5.85	8.25
VIRGINIA	VA09W-75	131.00	106.05	4.50	31.64	80.00	24.47	2.44	8.15
VIRGINIA	VA10W-119	128.50	79.38	6.25	35.92	92.50	33.44	9.75	18.93
VIRGINIA	VA10W-123	125.50	76.20	5.25	39.16	77.50	32.50	10.52	16.98
VIRGINIA	VA10W-125	123.75	82.55	6.50	37.39	80.00	29.53	14.18	26.35
VIRGINIA	VA10W-140	124.25	78.74	7.00	46.42	73.75	37.78	12.59	19.60
VIRGINIA	VA10W-21	126.75	76.84	6.50	47.12	90.00	44.40	11.54	19.70
VIRGINIA	VA10W-28	123.00	74.93	4.50	24.13	65.00	15.24	4.64	7.98
VIRGINIA	VA10W-663	127.25	85.09	5.00	25.26	76.25	19.80	4.16	9.08

[1]HD, heading date (Julian date); [2]PH, plant height (cm); [3]Rating, FHB rating (0 to 9); [4]SEV, severity (%); [5]INC, incidence (%); [6]Index (%); [7]FDK, Fusarium damaged kernel (%); [8]DON, deoxynivalenol (ppm).

Supplemental Table S3.2. SNP alleles for the markers detected for Fusarium damaged kernels and deoxynivalenol in the GWAS analyses (M9432, M6959 and M11423) for 250 soft red winter wheat lines grown in 2015 and 2016, Lexington, KY.

Name	M9432	M6959	M11423
011007A1-14-16-50	TT	TT	TT
0175A1-37-4-1	AA	AA	AA
02444A1-23-1-3	TT	TT	AA
03207A1-7-3-1	TT	AA	TT
03549A1-18-25	AA	AA	TT
03633A1-69-2-5	AA	AA	AT
04606RA1-1-7-1	AA	TT	TT
04606RA1-1-7-1-6	AA	TT	TT
04620A1-1-7-4	TT	TT	TT
04702A1-18	AA	TT	TT
04719A1-16-1-1-7	TT	TT	TT
0513A1-1-3	TT	TT	TT
05222A1-1-2-1	AA	TT	TT
05247A1-7-3-120	TT	TT	TT
05247A1-7-3-27	TT	AA	TT
05247A1-7-7-3-1	TT	TT	TT
05251A1-1-136-9-5	TT	TT	TT
05264A1-1-3-2	AA	TT	AA
05287A1-1-13	AA	AA	TT
0537A1-12	TT	TT	TT
0537A1-3-12	TT	TT	TT
0566A1-3-1-65	AA	TT	TT
0566A1-3-1-67	AA	TT	TT
0570A1-2-39-5	AA	AA	TT
06403A1-4	AA	TT	TT
0722A1-1-7	AA	TT	AA
07287RA1-14	TT	TT	TT
07290A1-12	AA	AT	TT
0762A1-2-8	AA	TT	AA
91193	TT	TT	TT
92201	TT	AA	TT
9346A1-2-5-5-2-1	TT	TT	TT
BECKER	AA	AA	TT
BESS	TT	AA	TT
BROMFIELD	TT	AA	TT

Supplemental Table S3.2. Continued

Name	M9432	M6959	M11423
CALEDONIA	TT	TT	AA
CATOCIN	TT	AT	TT
CAYUGA	TT	TT	AA
CLARK	TT	TT	TT
CRYSTAL	AA	AA	AA
D6234	TT	TT	AA
D8006	TT	TT	AA
E2041	AA	AA	AA
E5011	TT	AA	AA
E5024	TT	TT	AA
E6012	TT	TT	AA
ERNIE	TT	AA	TT
HOPKINS	TT	TT	AA
IL00-8109	TT	TT	TT
IL00-8530	AA	TT	TT
IL00-8633	AA	TT	TT
IL01-11934	TT	TT	TT
IL02-18228	AA	TT	TT
IL02-19483B	TT	TT	TT
IL04-24668	TT	TT	TT
IL04-9942	TT	TT	TT
IL05-4236	AA	AA	TT
IL06-13072	TT	TT	TT
IL06-13708	TT	TT	TT
IL06-13721	AA	AA	TT
IL06-14262	TT	TT	TT
IL06-14325	TT	TT	TT
IL06-23571	AA	TT	TT
IL06-31053	TT	TT	TT
IL06-7550	TT	TT	TT
IL06-7653	TT	TT	TT
IL07-12948	TT	TT	TT
IL07-16075	TT	TT	TT
IL07-19334	TT	TT	TT
IL07-20728	AA	TT	TT
IL07-20743	TT	TT	TT
IL07-21847	AA	TT	TT

Supplemental Table S3.2. Continued

Name	M9432	M6959	M11423
IL07-23420	AA	TT	TT
IL07-24841	AA	TT	TT
IL07-4415	AA	TT	TT
IL07-6861	TT	TT	TT
IL08-12174	TT	TT	TT
IL08-12206	AA	TT	TT
IL08-22075	TT	TT	TT
IL08-31639	AA	TT	TT
IL08-33373	AA	AA	TT
IL08-33951	TT	TT	TT
IL08-34020	TT	TT	TT
IL08-9266	TT	TT	TT
IL99-26442	TT	TT	TT
INW0411	AA	TT	AA
INW0412	TT	AA	TT
INW1021	AA	AA	TT
JAYPEE	TT	AA	TT
KY02C-1058-03	TT	TT	TT
KY02C-1076-07	TT	TT	TT
KY02C-1121-75	AA	TT	TT
KY02C-1122-06	TT	TT	AA
KY02C-2215-02	TT	TT	TT
KY03C-1002-02	TT	TT	TT
KY03C-1192-37	TT	TT	TT
KY03C-1195-10-1-5	TT	TT	TT
KY03C-1221-01	TT	TT	TT
KY03C-1221-06	AA	TT	TT
KY03C-1221-22	TT	TT	TT
KY03C-1237-01	TT	TT	TT
KY03C-1237-32	TT	TT	TT
KY03C-2047-02	TT	TT	TT
KY03C-2047-06	TT	TT	TT
KY03C-2049-02	TT	AA	TT
KY03C-2314-08	TT	TT	TT
KY03C-2399-02	TT	TT	TT
KY04C-1128-38-1-5	TT	TT	TT
KY04C-2006-41-1-1	AA	TT	TT
KY04C-2151-40	TT	TT	AA

Supplemental Table S3.2. Continued

Name	M9432	M6959	M11423
KY04C-2151-41	AA	TT	AA
KY04C-3006-33-14-3	TT	TT	AA
KY05C-1007-2-12-5	TT	TT	TT
KY05C-1105-42-20-1	TT	TT	TT
KY05C-1381-77-7-5	TT	TT	TT
KY05C-1617-17-17-3	TT	TT	AA
KY06C-1003-139-8-3	TT	AA	AA
KY93C-1238-17-1	AA	TT	TT
MALABAR	TT	AA	TT
MD01W270-10-3	TT	TT	TT
MD03W104-10-2	AA	AA	TT
MD03W151-10-12	TT	TT	TT
MD03W485-10-10	TT	TT	TT
MD03W485-10-12	TT	TT	TT
MD03W485-10-2	TT	TT	TT
MD03W485-10-8	TT	TT	TT
MD03W61-11-2	TT	TT	TT
MD03W61-11-3	TT	TT	AT
MD03W64-10-3	AA	TT	TT
MD03W665-10-3	TT	TT	TT
MD03W665-10-5	TT	TT	TT
MD04W1197-11-13	AA	TT	TT
MD04W249-11-12	TT	TT	TT
MD04W249-11-13	AA	TT	TT
MD04W249-11-5	AA	TT	TT
MD04W249-11-7	TT	TT	TT
MD04W359-11-10	AA	TT	TT
MD05W10208-11-13	TT	TT	TT
MD05W10208-11-14	TT	TT	TT
MD05W10208-11-3	TT	TT	TT
MD05W10208-11-6	TT	TT	TT
MD05W10208-11-7	TT	AA	TT
MD05W10208-11-8	TT	TT	TT
MD05W1292-11-1	TT	TT	TT
MD05W1292-11-4	TT	AA	TT
MD05W1317-11-4	TT	TT	TT
MD05W479-B-11-3	AA	TT	TT
MD07W272-11-5	TT	TT	TT

Supplemental Table S3.2. Continued

Name	M9432	M6959	M11423
MD07W419UM5-11-11	AA	AA	TT
MD07W419UM5-11-12	AA	AA	TT
MD665-09-6	AA	TT	TT
MEDINA	TT	TT	AA
MERL	AA	TT	TT
MO050921	TT	AA	TT
MO080103	TT	TT	TT
MO080104	TT	TT	TT
MO080584	TT	AA	TT
MO080589	TT	AA	TT
MO080864	TT	AA	TT
MO081163	TT	AA	TT
MO081280	TT	AA	TT
MO081559	TT	TT	TT
MO081652	TT	TT	TT
MO081699	TT	TT	TT
MO090574	TT	TT	TT
MO090581	TT	AA	TT
MO090821	TT	TT	TT
MO091011	AA	AA	TT
MO091159	AA	TT	TT
MO100172	TT	AA	TT
MO100231	TT	TT	TT
MO100265	TT	AA	TT
MO100519	TT	TT	TT
MO100535	TT	AA	TT
MO100539	TT	TT	TT
MO100647	AA	TT	TT
MO100745	TT	AA	TT
MO101329	TT	AA	TT
MO101358	TT	AA	TT
MO101361	TT	AA	TT
NY103-208-7263	TT	TT	AA
NY91017-8080	TT	TT	AA
NY96009-3037	TT	TT	AA
NY99066-3444	TT	TT	AA
OH05-200-74	TT	AA	TT
OH06-150-57	AA	TT	TT

Supplemental Table S3.2. Continued

Name	M9432	M6959	M11423
OH06-180-57	TT	TT	TT
OH07-166-41	TT	TT	TT
OH07-166-49	TT	TT	TT
OH07-174-11	AA	TT	TT
OH07-238-15	TT	TT	TT
OH07-254-11	TT	TT	TT
OH07-263-3	TT	TT	TT
OH07-94-70	TT	TT	TT
OH07-95-7	TT	TT	TT
OH07-98-21	TT	TT	TT
OH08-101-57	TT	AA	TT
OH08-101-72	AA	AA	TT
OH08-107-16	TT	AA	AA
OH08-133-25	AA	TT	TT
OH08-141-6	AA	TT	TT
OH08-149-11	TT	AA	TT
OH08-161-4	TT	TT	AA
OH08-161-78	TT	TT	AA
OH08-170-66	TT	TT	TT
OH08-172-42	TT	AA	TT
OH08-178-52	TT	TT	TT
OH08-180-48	TT	TT	TT
OH08-182-4	TT	TT	TT
OH08-199-1	TT	AA	TT
OH08-206-19	AA	TT	TT
OH08-207-33	TT	TT	AA
OH08-234-4	AA	TT	TT
OH08-235-33	AA	TT	TT
OH08-246-15	AA	TT	TT
OH08-256-47	AA	TT	TT
OH08-265-37	TT	TT	TT
OH08-269-58	AA	TT	TT
OH08-98-13	TT	AA	TT
PEMBROKE	TT	TT	AA
PIO 25R26	TT	TT	AA
REDRUBY	AA	TT	AA
SHIRLEY	TT	TT	TT
SS520	TT	AA	TT

Supplemental Table S3.2. Continued

Name	M9432	M6959	M11423
SS5205	TT	AA	TT
SSMPV57	AA	TT	TT
USG3315	TT	TT	TT
VA05W-151	TT	AA	TT
VA05W-251	TT	TT	AA
VA07W-415	TT	AA	TT
VA08MAS-369	TT	TT	TT
VA08W-176	TT	TT	TT
VA08W-294	AA	AA	TT
VA08W-613	AA	TT	TT
VA09W-110	TT	TT	TT
VA09W-112	TT	TT	TT
VA09W-114	TT	TT	TT
VA09W-188WS	TT	TT	TT
VA09W-46	AA	AA	TT
VA09W-52	TT	AA	TT
VA09W-69	AA	AA	TT
VA09W-73	AA	AA	TT
VA09W-75	AA	AA	TT
VA10W-119	AA	AA	TT
VA10W-123	AA	AA	TT
VA10W-125	AA	AA	TT
VA10W-140	TT	TT	TT
VA10W-21	TT	TT	TT
VA10W-28	TT	TT	TT
VA10W-663	TT	TT	TT

Supplemental Table S3.3. SNP information for markers detected in the GWAS analysis for 250 soft red winter wheat lines grown in 2015 and 2016, Lexington, KY.

Trait	Year	SNP	SNP id	SNP Name	Chr.[1]	cM[2]	Rescaled distance (cM)	MAF[3]	FDR_Adjustes_P-values[4]
PH[5]	Average	M10186	IWA5898	wsnp_JD_c19278_17450072	6A	190.27	79.07515	0.112	0.966946
	2015	.	.	.	.	.	.	.	.
	2016	M11214	IWB73456	Tdurum_contig7981_70	6B	245.09	71.76409	0.444	0.991595
RAT.[6]	Average	M12955*	IWB41483	Kukri_c15912_2330	7B	359.99	112.44194	0.13	0.923164
		M12960*	IWB11413	BS00085556_51	7B	379.94	118.67327	0.142	0.923164
		M12143	IWB10812	BS00075525_51	7A	398.23	135.61603	0.152	0.923164
	2015	.	.	.	.	.	.	.	.
	2016	M1458	IWB1335	BobWhite_c20015_225	1B	238.2	74.37157	0.404	0.597162
		M7852	IWB10092	BS00067606_51	5A	79.39	15.85636	0.268	0.597162
		M1318	IWB3214	BobWhite_c42170_144	1B	7.67	3.46314	0.486	0.597162
		M1461	IWB10630	BS00072982_51	1B	238.2	74.37157	0.306	0.597162
SEV[7]	Average	.	.	.	.	.	.	.	.
	2015	M9385*	IWB25279	Excalibur_c32979_1152	5B	433.05	139.39594	0.152	0.396903
		M5248*	IWA1998	wsnp_Ex_c15269_23491104	3A	284.21	89.22548	0.282	0.575510
		M2347	IWB12340	BS00107935_51	1D	435.16	140.92900	0.412	0.594565
		M1657	IWA2889	wsnp_Ex_c24318_33561600	1B	269.73	84.43308	0.13	0.594565
		M654	IWB31790	GENE-0293_346	1A	440.55	110.15332	0.494	0.594565
	2016	M6781	IWB26155	Excalibur_c40618_182	4A	147.89	37.81507	0.124	0.200695
		M11507	IWB41216	Kukri_c14511_1046	6B	410.92	120.32029	0.342	0.465363
		M12936	IWB56849	RAC875_c33564_454	7B	321.13	100.30412	0.294	0.465363
		M11498	IWB9630	BS00065783_51	7B	408.89	119.72590	0.398	0.817158
INC[8]	Average	M12955*	IWB41483	Kukri_c15912_2330	7B	359.99	112.44194	0.13	0.305976

[1]Chr., Chromosome; [2]cM, centimorgan; [3]MAF, minor allele frequency; [4]FDR_Adjusted_P-values, false discovery rate adjusted p-value. [5]PH, plant height (cm); [6]RAT, rating (0 to 9); [7]SEV, severity (%); [8]INC, incidence (%); [9]Index (%); [10]FDK, Fusarium damaged kernel (%); [11]DON, deoxynivalenol (ppm).
*, markers with pleotropic effect

Supplemental Table S3.3. Continued

Trait	Year	SNP	SNP id	SNP Name	Chr.	cM	Rescaled distance (cM)	MAF	FDR_Adjustes_P-values
INC[8]	Average	M12960*	IWB11413	BS00085556_51	7B	379.94	118.67327	0.142	0.305976
		M5559	IWB11738	BS00091887_51	3A	433.76	136.17553	0.23	0.496772
		M12957*	IWB71789	Tdurum_contig47317_100	7B	362.32	113.16971	0.172	0.496772
		M5589	IWA94	wsnp_BE426418A_Ta_2_1	3A	513.89	161.33171	0.25	0.496772
	2015	M3268	IWA2571	wsnp_Ex_c21092_30220342	2B	240.19	74.89946	0.328	0.765553
		M12960*	IWB11413	BS00085556_51	7B	379.94	118.67327	0.142	0.793384
	2016	M7272	IWB931	BobWhite_c1656_186	4B	71.97	24.66584	0.408	0.156804
		M7271	IWB932	BobWhite_c1656_845	4B	63.55	21.78010	0.318	0.522987
		M2050	IWB55046	RAC875_c2070_566	1D	5.47	3.40225	0.172	0.522987
Index[9]	Average	M12955*	IWB41483	Kukri_c15912_2330	7B	359.99	112.44194	0.13	0.952809
		M12957*	IWB71789	Tdurum_contig47317_100	7B	362.32	113.16971	0.172	0.952809
		M12960*	IWB11413	BS00085556_51	7B	379.94	118.67327	0.142	0.952809
	2015	M9385*	IWB25279	Excalibur_c32979_1152	5B	433.05	139.39594	0.152	0.582600
		M5248*	IWA1998	wsnp_Ex_c15269_23491104	3A	284.21	89.22548	0.282	0.582600
		M3268*	IWA2571	wsnp_Ex_c21092_30220342	2B	240.19	74.89946	0.328	0.582600
		M7759	IWB73485	Tdurum_contig81113_395	4B	327.27	112.16325	0.462	0.811515
	2016	.	.	.	.	.	.	.	.
FDK[10]	Average	M9432*	IWB45354	Kukri_c4349_639	5B	447.74	144.12455	0.304	0.993211
		M11423	IWA3268	wsnp_Ex_c3025_5587183	6B	375.21	109.86415	0.144	0.993211
	2015	M2926	IWB59335	RAC875_c58006_492	2A	413.64	123.64531	0.264	0.666727
		M9385*	IWB25279	Excalibur_c32979_1152	5B	433.05	139.39594	0.152	0.959209
	2016	M11046	IWB59754	RAC875_c63209_154	6B	226.64	66.36180	0.388	0.980406
		M2977	IWB51957	Ra_c3750_1082	2A	473.89	141.65525	0.164	0.980406
DON[11]	Average	M9432*	IWB45354	Kukri_c4349_630	5B	447.74	144.12455	0.304	0.065466

[1]Chr., Chromosome; [2]cM, centimorgan; [3]MAF, minor allele frequency; [4]FDR_Adjusted_P-values, false discovery rate adjusted p-value. [5]PH, plant height (cm); [6]RAT, rating (0 to 9); [7]SEV, severity (%); [8]INC, incidence (%); [9]Index (%); [10]FDK, Fusarium damaged kernel (%); [11]DON, deoxynivalenol (ppm).
*, markers with pleotropic effect

Supplemental Table S3.3. Continued

Trait	Year	SNP	SNP id	SNP Name	Chr.	cM	Rescaled distance (cM)	MAF	FDR_Adjustes_P-values
DON[11]	Average	M6959	IWB60934	RAC875_c88582_131	4A	356.78	91.22768	0.264	0.952796
	2015	.	.	.	.	.	.	.	.
	2016	M13271	IWB35552	IAAV8855	7D	337.29	152.29241	0.112	0.431617
		M9432*	IWB45354	Kukri_c4349_639	5B	447.74	144.12455	0.304	0.610326

[1]Chr., Chromosome; [2]cM, centimorgan; [3]MAF, minor allele frequency; [4]FDR_Adjusted_P-values, false discovery rate adjusted p-value. [5]PH, plant height (cm); [6]RAT, rating (0 to 9); [7]SEV, severity (%); [8]INC, incidence (%); [9]Index (%); [10]FDK, Fusarium damaged kernel (%); [11]DON, deoxynivalenol (ppm).
*, markers with pleotropic effect

Supplemental Table S3.4. Quantitative trait locus (QTL) effect on severity (SEV), incidence (INC) and index means of 250 soft red winter wheat lines grown in 2015 and 2016 in Lexington, KY.

QTL[1]	Number of lines	SEV		INC		INDEX	
no Fhb1[2]	229	29.85	A	78.41	A	24.31	A
Fhb1[3]	19	29.61	A	79.61	A	24.09	A
Rht-B1a[4]	121	31.28	A	79.34	A	25.72	A
Rht-B1b[5]	125	28.43	B	77.89	A	22.97	B
Rht-D1a[6]	157	28.32	A	77.73	B	22.9	B
Rht-D1b[7]	88	32.66	B	80.31	A	27.02	A
vrn-A1[8]	236	29.78	A	78.59	A	24.27	A
Vrn-A1-short[9]	14	29.94	A	77.14	A	24.09	A
Vrn-B1[10]	242	29.66	A	78.4	A	24.14	A
Vrn-B1-short[11]	5	33.61	A	79.5	A	26.83	A
Vrn-D3b[12]	171	30.71	A	79.1	A	25.07	A
Vrn-D3a-early[13]	76	27.89	B	77.31	A	22.65	B
Ppd-A1a[14]	140	28.73	B	77.88	A	23.2	B
Ppd-A1b[15]	104	31.29	A	79.44	A	25.77	A
Ppd-B1a[16]	19	33.54	A	78.29	A	27.74	A
Ppd-B1b[17]	188	29.34	B	78.44	A	23.81	A
Ppd-D1a[18]	123	30.53	A	79.1	A	24.88	A
Ppd-D1b[19]	123	29.11	A	78.17	A	23.76	A

[1]QTL, quantitative trait loci; [2]*Fhb1*-S, susceptible; [3]*Fhb1*-R, resistant; [4]*Rht-B1a* and [6]*Rht-D1a*, height wild type allele; [5]*Rht-B1b* and [7]*Rht-D1b*, dwarfing height allele; [8]*Vrn-A1*, [10]*Vrn-B1* and [12]*Vrn-D3*, vernalization wild type allele; [9]V*rn-A1-short*, [11]*Vrn-B1-short* and [13]*Vrn-D3-early* mutant allele; [14]*Ppd-A1a*, [16]*Ppd-B1a* and [18]*Ppd-D1a*, photoperiod insensitive; [15]*Ppd-A1b*, [17]*Ppd-B1b* and [19]*Ppd-D1b,* photoperiod sensitive. Within columns, means followed by the same letter are not significantly different according to *t* test (0.05).

Supplemental Table S3.5. Quantitative trait locus (QTL) for Rht-B1, Rht-D1, Vrn-A1, Vrn-B1 and Vrn-D3 in 256 soft red winter wheat lines grown in 2015 and 2016, Lexington, KY.

Name	*Rht-B1*	*Rht-D1*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3*
011007A1-14-16-50	*Rht-B1b het*	*Rht-D1b het*	*Vrn-A1*[3]	*Vrn-B1*[3]	*Vrn-D3a_early*[4]
0175A1-37-4-1	*Rht-B1b*[2]	*Rht-D1a*[1]	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*[3]
02444A1-23-1-3	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
03207A1-7-3-1	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
03549A1-18-25	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
03633A1-69-2-5	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
04606RA1-1-7-1	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
04606RA1-1-7-1-6	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
04620A1-1-7-4	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
04702A1-18	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
04719A1-16-1-1-7	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
0513A1-1-3	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
05222A1-1-2-1	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
05247A1-7-3-120	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
05247A1-7-3-27	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
05247A1-7-7-3-1	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
05251A1-1-136-9-5	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
05264A1-1-3-2	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
05287A1-1-13	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
0537A1-12	*Rht-B1a*[1]	*Rht-D1b*[2]	*Vrn-A1*	*Vrn-B1_short*[4]	*Vrn-D3b*
0537A1-3-12	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1_short*	*Vrn-D3b*
0566A1-3-1-65	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
0566A1-3-1-67	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
0570A1-2-39-5	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
06403A1-4	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
0722A1-1-7	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
07287RA1-14	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
07290A1-12	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
0762A1-2-8	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
91193	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early het*
92201	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
9346A1-2-5-5-2-1	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
BECKER	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
BESS	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
BROMFIELD	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*

[1]*Rht-B1a* and *Rht-D1a*, height wild type allele; [2]*Rht-B1b* and *Rht-D1b*, dwarfing height allele; [3]*Vrn-A1*, *Vrn-B1* and *Vrn-D3*, vernalization wild type allele; [4]V*rn-A1-short*, *Vrn-B1-short* and *Vrn-D3-early*, mutant allele.

Supplemental Table S3.5. Continued

Name	*Rht-B1*	*Rht-D1*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3*
CALEDONIA	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
CATOCIN	*Rht-B1b het*	*Rht-D1b het*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
CAYUGA	*Rht-B1a*	*Rht-D1b het*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
CHOPTANK	*Rht-B1b*	*no call*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
CLARK	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
CRYSTAL	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
D6234	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
D8006	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
E2041	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
E5011	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
E5024	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
E6012	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
ERNIE	*Rht-B1b*	*Rht-D1a*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3a_early*
FOSTER	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
HOPKINS	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL00-8109	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL00-8530	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL00-8633	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL01-11934	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early het*
IL02-18228	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL02-19483B	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL04-24668	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL04-9942	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL05-4236	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL06-13072	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL06-13708	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL06-13721	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL06-14262	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL06-14325	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL06-23571	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL06-31053	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL06-7550	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL06-7653	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1_short*	*Vrn-D3b*
IL07-12948	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL07-16075	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*null?*	*Vrn-D3a_early*
IL07-19334	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL07-20728	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL07-20743	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*

[1]*Rht-B1a* and *Rht-D1a*, height wild type allele; [2]*Rht-B1b* and *Rht-D1b*, dwarfing height allele; [3]*Vrn-A1*, *Vrn-B1* and *Vrn-D3*, vernalization wild type allele; [4]V*rn-A1-short*, *Vrn-B1-short* and *Vrn-D3-early*, mutant allele.

Supplemental Table S3.5. Continued

Name	*Rht-B1*	*Rht-D1*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3*
IL07-21847	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL07-23420	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL07-24841	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL07-4415	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL07-6861	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL08-12174	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL08-12206	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL08-22075	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL08-31639	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
IL08-33373	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL08-33951	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL08-34020	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL08-9266	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
IL99-26442	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
INW0411	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
INW0412	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
INW1021	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
JAYPEE	*Rht-B1a*	*Rht-D1b*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3b*
KY02C-1058-03	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY02C-1076-07	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY02C-1121-75	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY02C-1122-06	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY02C-2215-02	*Rht-B1a*	*Rht-D1a*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3b*
KY03C-1002-02	*Rht-B1b het*	*Rht-D1b het*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
KY03C-1192-37	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY03C-1195-10-1-5	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
KY03C-1221-01	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY03C-1221-06	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY03C-1221-22	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY03C-1237-01	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY03C-1237-32	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
KY03C-2047-02	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY03C-2047-06	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY03C-2049-02	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY03C-2314-08	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY03C-2399-02	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY04C-1128-38-1-5	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY04C-2006-41-1-1	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*

[1]*Rht-B1a* and *Rht-D1a*, height wild type allele; [2]*Rht-B1b* and *Rht-D1b*, dwarfing height allele; [3]*Vrn-A1*, *Vrn-B1* and *Vrn-D3*, vernalization wild type allele; [4]V*rn-A1-short*, *Vrn-B1-short* and *Vrn-D3-early*, mutant allele.

Supplemental Table S3.5. Continued

Name	*Rht-B1*	*Rht-D1*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3*
KY04C-2151-40	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY04C-2151-41	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
KY04C-3006-33-14-3	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY05C-1007-2-12-5	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
KY05C-1105-42-20-1	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY05C-1381-77-7-5	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY05C-1617-17-17-3	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY06C-1003-139-8-3	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
KY93C-1238-17-1	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MALABAR	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MD01W270-10-3	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W104-10-2	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W151-10-12	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W485-10-10	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W485-10-12	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W485-10-2	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W485-10-8	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W61-11-2	*Rht-B1a*	*Rht-D1b*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3b*
MD03W61-11-3	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W64-10-3	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W665-10-3	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD03W665-10-5	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD04W1197-11-13	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD04W249-11-12	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD04W249-11-13	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD04W249-11-5	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD04W249-11-7	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD04W359-11-10	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD04W8-11-4	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD05W10208-11-13	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD05W10208-11-14	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD05W10208-11-3	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD05W10208-11-6	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD05W10208-11-7	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MD05W10208-11-8	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD05W1292-11-1	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD05W1292-11-4	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD05W1317-11-4	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*

[1]*Rht-B1a* and *Rht-D1a*, height wild type allele; [2]*Rht-B1b* and *Rht-D1b*, dwarfing height allele; [3]*Vrn-A1*, *Vrn-B1* and *Vrn-D3*, vernalization wild type allele; [4]V*rn-A1-short*, *Vrn-B1-short* and *Vrn-D3-early*, mutant allele.

Supplemental Table S3.5. Continued

Name	*Rht-B1*	*Rht-D1*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3*
MD05W479-B-11-3	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD07W272-11-5	*Rht-B1a*	*Rht-D1b*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3b*
MD07W419UM5-11-11	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD07W419UM5-11-12	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MD665-09-6	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MEDINA	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MERL	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO050921	*Rht-B1b*	*Rht-D1a*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3a_early*
MO080103	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO080104	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO080584	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO080589	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO080864	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO081163	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO081280	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO081559	*Rht-B1b*	*Rht-D1a*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3a_early*
MO081652	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO081699	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO090574	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO090581	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO090821	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO091011	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early het*
MO091159	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO100172	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO100231	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO100265	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO100519	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO100535	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO100539	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO100647	*Rht-B1b het*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO100745	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
MO101329	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO101358	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
MO101361	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
NY103-208-7263	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
NY91017-8080	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
NY96009-3037	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
NY99066-3444	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*

[1]*Rht-B1a* and *Rht-D1a*, height wild type allele; [2]*Rht-B1b* and *Rht-D1b*, dwarfing height allele; [3]*Vrn-A1*, *Vrn-B1* and *Vrn-D3*, vernalization wild type allele; [4]V*rn-A1-short*, *Vrn-B1-short* and *Vrn-D3-early*, mutant allele.

Supplemental Table S3.5. Continued

Name	*Rht-B1*	*Rht-D1*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3*
OH05-200-74	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH06-150-57	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH06-180-57	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH07-166-41	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH07-166-49	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH07-174-11	*Rht-B1b*	*Rht-D1a*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3b*
OH07-238-15	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH06-180-57	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH07-254-11	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH07-263-3	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH07-94-70	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH07-95-7	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH07-98-21	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-101-57	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-101-72	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-107-16	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-133-25	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-141-6	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-149-11	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-161-4	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-161-78	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-170-66	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-172-42	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-178-52	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-180-48	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-182-4	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-199-1	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-206-19	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-207-33	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-234-4	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-235-33	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-246-15	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-254-22	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-256-47	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
OH08-265-37	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-269-58	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
OH08-98-13	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
PEMBROKE	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1_short*	*Vrn-D3b*

[1]*Rht-B1a* and *Rht-D1a*, height wild type allele; [2]*Rht-B1b* and *Rht-D1b*, dwarfing height allele; [3]*Vrn-A1*, *Vrn-B1* and *Vrn-D3*, vernalization wild type allele; [4]V*rn-A1-short*, *Vrn-B1-short* and *Vrn-D3-early*, mutant allele.

Supplemental Table S3.5. Continued

Name	*Rht-B1*	*Rht-D1*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3*
PIO 25R26	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
REDRUBY	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
SHIRLEY	*Rht-B1b*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
SS520	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
SS5205	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3a_early*
SSMPV57	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
TRUMAN	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
USG3315	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1_short*	*Vrn-D3b*
VA05W-151	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA05W-251	*Rht-B1a*	*Rht-D1b*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3b*
VA06W-412	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA07W-415	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA08MAS-369	*Rht-B1a*	*Rht-D1b*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3a_early*
VA08W-176	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA08W-294	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA08W-613	*Rht-B1a*	*Rht-D1b*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3a_early*
VA09W-110	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA09W-112	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA09W-114	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA09W-188WS	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA09W-46	*Rht-B1a*	*Rht-D1b*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3b*
VA09W-52	*Rht-B1a*	*Rht-D1b*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3b*
VA09W-69	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*null?*	*Vrn-D3b*
VA09W-73	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA09W-75	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA10W-119	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA10W-123	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA10W-125	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1_short het*	*Vrn-D3b*
VA10W-140	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA10W-21	*Rht-B1a*	*Rht-D1b*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA10W-28	*Rht-B1a*	*Rht-D1a*	*Vrn-A1*	*Vrn-B1*	*Vrn-D3b*
VA10W-663	*Rht-B1b*	*Rht-D1a*	*Vrn-A1_short*	*Vrn-B1*	*Vrn-D3b*

[1]*Rht-B1a* and *Rht-D1a*, height wild type allele; [2]*Rht-B1b* and *Rht-D1b*, dwarfing height allele; [3]*Vrn-A1*, *Vrn-B1* and *Vrn-D3*, vernalization wild type allele; [4]V*rn-A1-short*, *Vrn-B1-short* and *Vrn-D3-early*, mutant allele.

Supplemental Table S3.6. Quantitative trait locus (QTL) for Ppd-A1, Ppd-B1, Ppd-D1 and Fhb in 256 soft red winter wheat lines grown in 2015 and 2016, Lexington, KY.

Name	*Ppd-A1*	*Ppd-B1*	*Ppd-D1*	*Fhb1*
011007A1-14-16-50	*Ppd-A1a.1_insens het*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens het*	*no*
0175A1-37-4-1	*Ppd-A1a.1_insens*[2]	*null*	*Ppd-D1a_insens*[2]	*Fhb1*
02444A1-23-1-3	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*[1]	*Ppd-D1a_insens*	*Fhb1*
03207A1-7-3-1	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*[1]	*no*
03549A1-18-25	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*Fhb1*
03633A1-69-2-5	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens het*	*Fhb1*
04606RA1-1-7-1	*Ppd-A1b_sens*[1]	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
04606RA1-1-7-1-6	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
04620A1-1-7-4	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*[2]	*Ppd-D1b_sens*	*no*
04702A1-18	*Ppd-A1a.1_insens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
04719A1-16-1-1-7	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
0513A1-1-3	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
05222A1-1-2-1	*Ppd-A1a.1_insens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
05247A1-7-3-120	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
05247A1-7-3-27	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
05247A1-7-7-3-1	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
05251A1-1-136-9-5	*Ppd-A1b_sens*	*Ppd-B1a_S64_insens*	*Ppd-D1b_sens*	*no*
05264A1-1-3-2	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
05287A1-1-13	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
0537A1-12	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
0537A1-3-12	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
0566A1-3-1-65	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
0566A1-3-1-67	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
0570A1-2-39-5	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
06403A1-4	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*Fhb1*
0722A1-1-7	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*Fhb1*
07287RA1-14	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
07290A1-12	*Ppd-A1a.1_insens het*	*Ppd-B1a_S64_insens*	*Ppd-D1a_insens*	*Fhb1*
0762A1-2-8	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*Fhb1*
91193	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
92201	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
9346A1-2-5-5-2-1	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
BECKER	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
BESS	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
BROMFIELD	*Ppd-A1a.1_insens het*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
CALEDONIA	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
CATOCIN	*Ppd-A1a.1_insens*	*Ppd-B1b_sens*	*Ppd-D1a_insens het*	*no*

[1]*Ppd-A1a*, *Ppd-B1a* and *Ppd-D1a*, photoperiod insensitive; [2]*Ppd-A1b*, *Ppd-B1b* and *Ppd-D1b,* photoperiod sensitive.

Supplemental Table S3.6. Continued

Name	*Ppd-A1*	*Ppd-B1*	*Ppd-D1*	*Fhb1*
CAYUGA	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
CHOPTANK	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
CLARK	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
CRYSTAL	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
D6234	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
D8006	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
E2041	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no call*
E5011	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
E5024	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
E6012	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
ERNIE	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
FOSTER	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
HOPKINS	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*Fhb1*
IL00-8109	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
IL00-8530	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
IL00-8633	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
IL01-11934	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL02-18228	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL02-19483B	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
IL04-24668	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
IL04-9942	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL05-4236	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL06-13072	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL06-13708	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL06-13721	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
IL06-14262	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
IL06-14325	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
IL06-23571	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
IL06-31053	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL06-7550	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL06-7653	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL07-12948	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
IL07-16075	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
IL07-19334	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL07-20728	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL07-20743	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL07-21847	*Ppd-A1b_sens*	*null*	*Ppd-D1b_sens*	*no*
IL07-23420	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*

[1]*Ppd-A1a*, *Ppd-B1a* and *Ppd-D1a*, photoperiod insensitive; [2]*Ppd-A1b*, *Ppd-B1b* and *Ppd-D1b,* photoperiod sensitive.

Supplemental Table S3.6. Continued

Name	*Ppd-A1*	*Ppd-B1*	*Ppd-D1*	*Fhb1*
IL07-24841	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL07-4415	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*Fhb1*
IL07-6861	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL08-12174	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
IL08-12206	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
IL08-22075	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
IL08-31639	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
IL08-33373	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL08-33951	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
IL08-34020	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
IL08-9266	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
IL99-26442	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
INW0411	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*Fhb1*
INW0412	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
INW1021	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*Fhb1*
JAYPEE	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
KY02C-1058-03	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY02C-1076-07	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY02C-1121-75	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
KY02C-1122-06	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY02C-2215-02	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY03C-1002-02	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens het*	*no*
KY03C-1192-37	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY03C-1195-10-1-5	*Ppd-A1b_sens*	*Ppd-B1a_S64_insens*	*Ppd-D1a_insens*	*no*
KY03C-1221-01	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY03C-1221-06	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY03C-1221-22	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*Fhb1*
KY03C-1237-01	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY03C-1237-32	*Ppd-A1b_sens*	*Ppd-B1a_S64_insens*	*Ppd-D1a_insens*	*Fhb1*
KY03C-2047-02	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
KY03C-2047-06	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY03C-2049-02	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY03C-2314-08	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
KY03C-2399-02	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
KY04C-1128-38-1-5	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
KY04C-2006-41-1-1	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
KY04C-2151-40	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*Fhb1*
KY04C-2151-41	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*Fhb1*

[1]*Ppd-A1a*, *Ppd-B1a* and *Ppd-D1a*, photoperiod insensitive; [2]*Ppd-A1b*, *Ppd-B1b* and *Ppd-D1b,* photoperiod sensitive.

Supplemental Table S3.6. Continued

Name	*Ppd-A1*	*Ppd-B1*	*Ppd-D1*	*Fhb1*
KY04C-3006-33-14-3	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY05C-1007-2-12-5	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
KY05C-1105-42-20-1	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY05C-1381-77-7-5	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY05C-1617-17-17-3	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
KY06C-1003-139-8-3	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
KY93C-1238-17-1	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MALABAR	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MD01W270-10-3	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
MD03W104-10-2	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MD03W151-10-12	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
MD03W485-10-10	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
MD03W485-10-12	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
MD03W485-10-2	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
MD03W485-10-8	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
MD03W61-11-2	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
MD03W61-11-3	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
MD03W64-10-3	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MD03W665-10-3	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
MD03W665-10-5	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
MD04W1197-11-13	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MD04W249-11-12	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
MD04W249-11-13	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MD04W249-11-5	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MD04W249-11-7	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MD04W359-11-10	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MD04W8-11-4	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MD05W10208-11-13	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MD05W10208-11-14	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
MD05W10208-11-3	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MD05W10208-11-6	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MD05W10208-11-7	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MD05W10208-11-8	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
MD05W1292-11-1	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
MD05W1292-11-4	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
MD05W1317-11-4	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
MD05W479-B-11-3	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*Fhb1*
MD07W272-11-5	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*Fhb1*
MD07W419UM5-11-11	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*

[1]*Ppd-A1a*, *Ppd-B1a* and *Ppd-D1a*, photoperiod insensitive; [2]*Ppd-A1b*, *Ppd-B1b* and *Ppd-D1b,* photoperiod sensitive.

Supplemental Table S3.6. Continued

Name	*Ppd-A1*	*Ppd-B1*	*Ppd-D1*	*Fhb1*
MD07W419UM5-11-12	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MD665-09-6	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
MEDINA	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MERL	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MO050921	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO080103	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO080104	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MO080584	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO080589	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO080864	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO081163	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO081280	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO081559	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO081652	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MO081699	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MO090574	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
MO090581	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO090821	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO091011	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO091159	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
MO100172	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO100231	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO100265	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*Fhb1*
MO100519	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO100535	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO100539	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO100647	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO100745	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO101329	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO101358	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
MO101361	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
NY103-208-7263	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
NY91017-8080	*Ppd-A1b_sens*	*Ppd-B1a_S64_insens*	*Ppd-D1b_sens*	*no*
NY96009-3037	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
NY99066-3444	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH05-200-74	*Ppd-A1b_sens*	*Ppd-B1a_S64_insens*	*Ppd-D1b_sens*	*no*
OH06-150-57	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
OH06-180-57	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
OH07-166-41	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*

[1]*Ppd-A1a*, *Ppd-B1a* and *Ppd-D1a*, photoperiod insensitive; [2]*Ppd-A1b*, *Ppd-B1b* and *Ppd-D1b,* photoperiod sensitive.

Supplemental Table S3.6. Continued

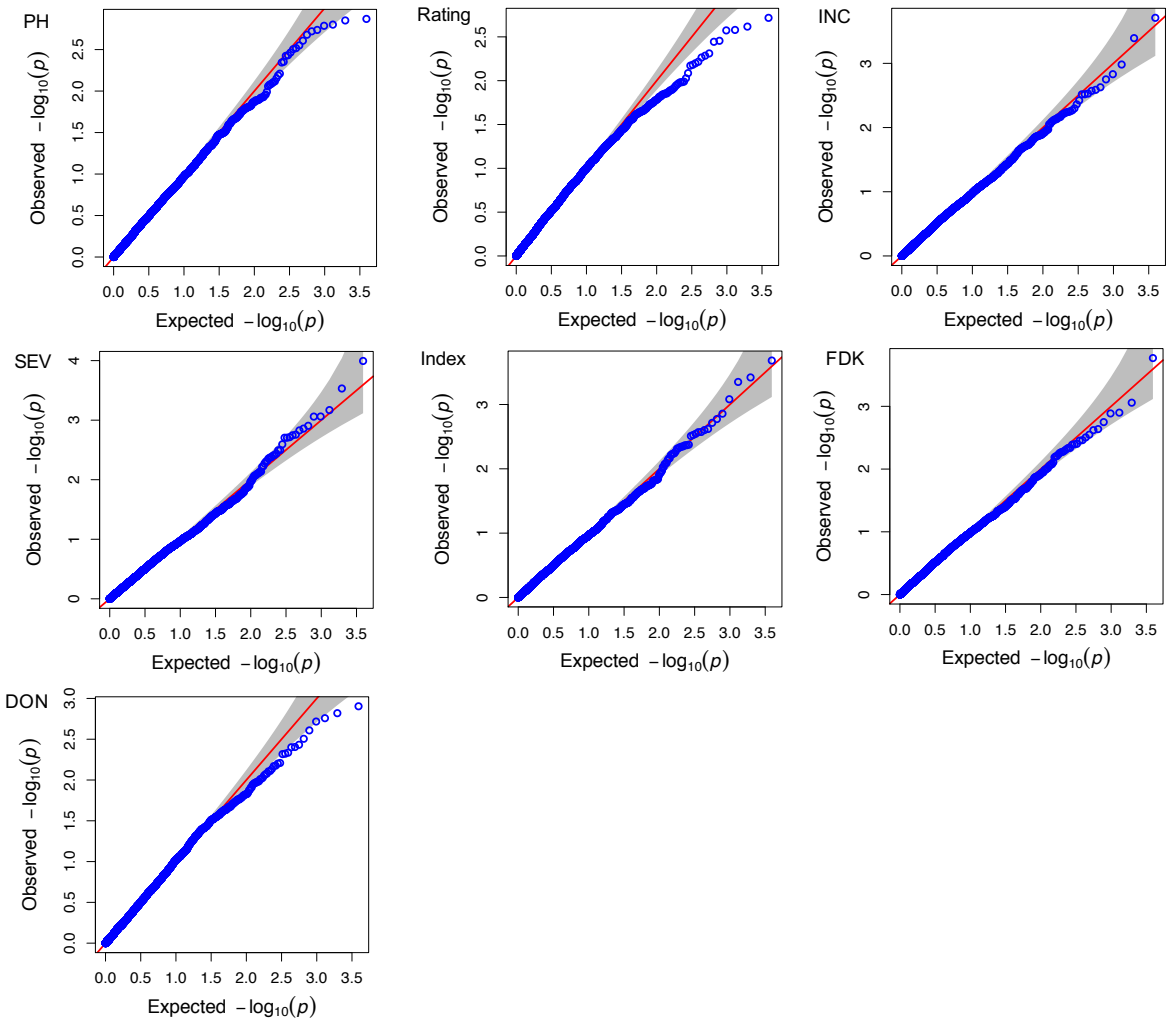
Name	*Ppd-A1*	*Ppd-B1*	*Ppd-D1*	*Fhb1*
OH07-166-49	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
OH07-174-11	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
OH07-238-15	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
OH07-254-11	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
OH07-263-3	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH07-94-70	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
OH07-95-7	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
OH07-98-21	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-101-57	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-101-72	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-107-16	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no call*
OH08-133-25	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
OH08-141-6	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-149-11	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
OH08-161-4	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
OH08-161-78	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
OH08-170-66	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-172-42	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-178-52	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-180-48	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-182-4	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
OH08-199-1	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-206-19	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-207-33	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1b_sens*	*no*
OH08-234-4	*Ppd-A1b_sens*	*null*	*Ppd-D1b_sens*	*no*
OH08-235-33	*Ppd-A1b_sens*	*null*	*Ppd-D1b_sens*	*no*
OH08-246-15	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-254-22	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-256-47	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-265-37	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
OH08-269-58	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
OH08-98-13	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
PEMBROKE	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
PIO 25R26	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
REDRUBY	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
SHIRLEY	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
SS520	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
SS5205	*Ppd-A1b_sens*	*Ppd-B1a_CS_insens*	*Ppd-D1a_insens*	*no*
SSMPV57	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*

[1]*Ppd-A1a*, *Ppd-B1a* and *Ppd-D1a*, photoperiod insensitive; [2]*Ppd-A1b*, *Ppd-B1b* and *Ppd-D1b,* photoperiod sensitive.

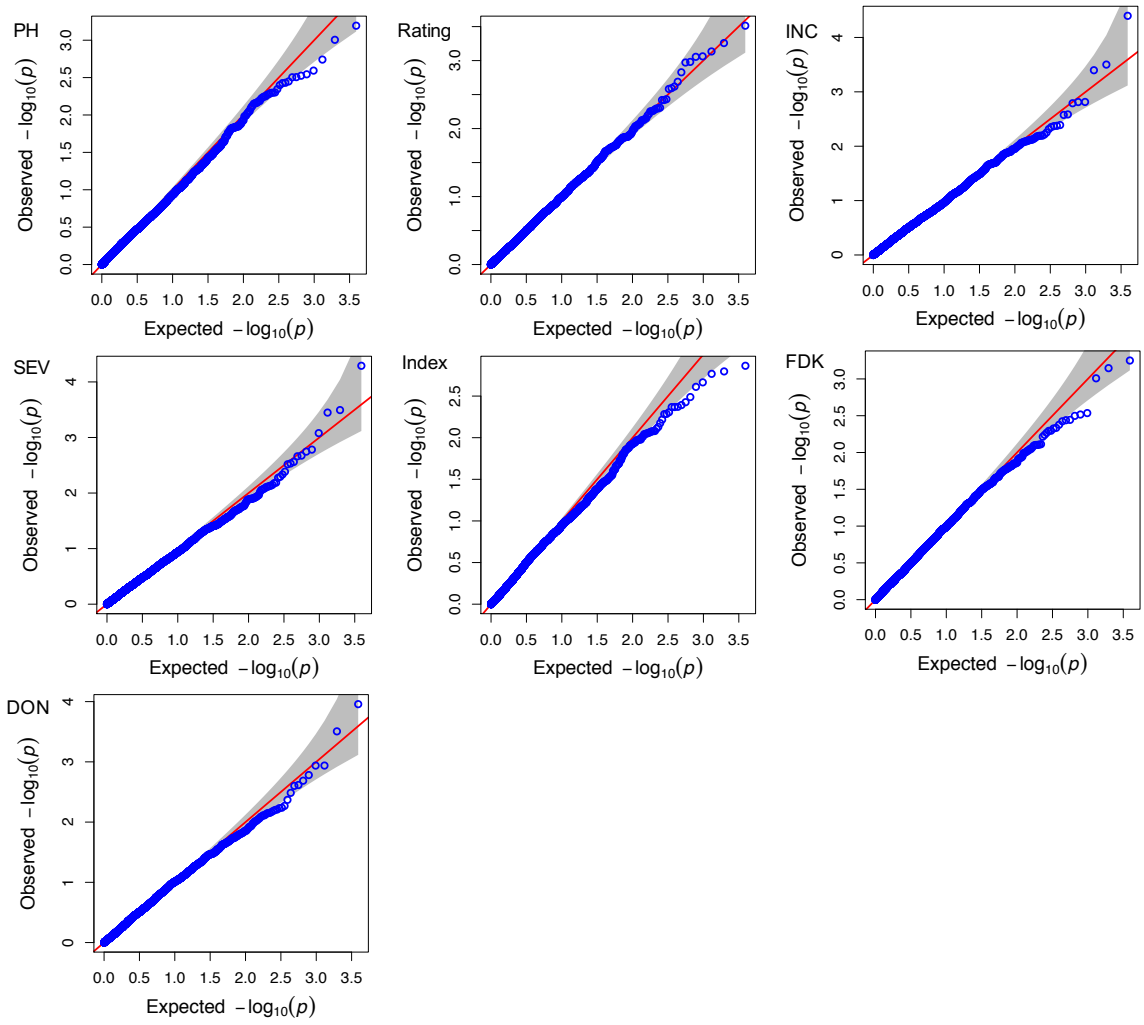
Supplemental Table S3.6. Continued

Name	*Ppd-A1*	*Ppd-B1*	*Ppd-D1*	*Fhb1*
TRUMAN	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
USG3315	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA05W-151	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
VA05W-251	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA06W-412	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
VA07W-415	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA08MAS-369	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
VA08W-176	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
VA08W-294	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
VA08W-613	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA09W-110	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
VA09W-112	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*
VA09W-114	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA09W-188WS	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA09W-46	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA09W-52	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA09W-69	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
VA09W-73	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
VA09W-75	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1b_sens*	*no*
VA10W-119	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA10W-123	*Ppd-A1b_sens*	*Ppd-B1a_S64_insens*	*Ppd-D1a_insens*	*no*
VA10W-125	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA10W-140	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA10W-21	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA10W-28	*Ppd-A1b_sens*	*Ppd-B1b_sens*	*Ppd-D1a_insens*	*no*
VA10W-663	*Ppd-A1b_sens*	*null*	*Ppd-D1a_insens*	*no*

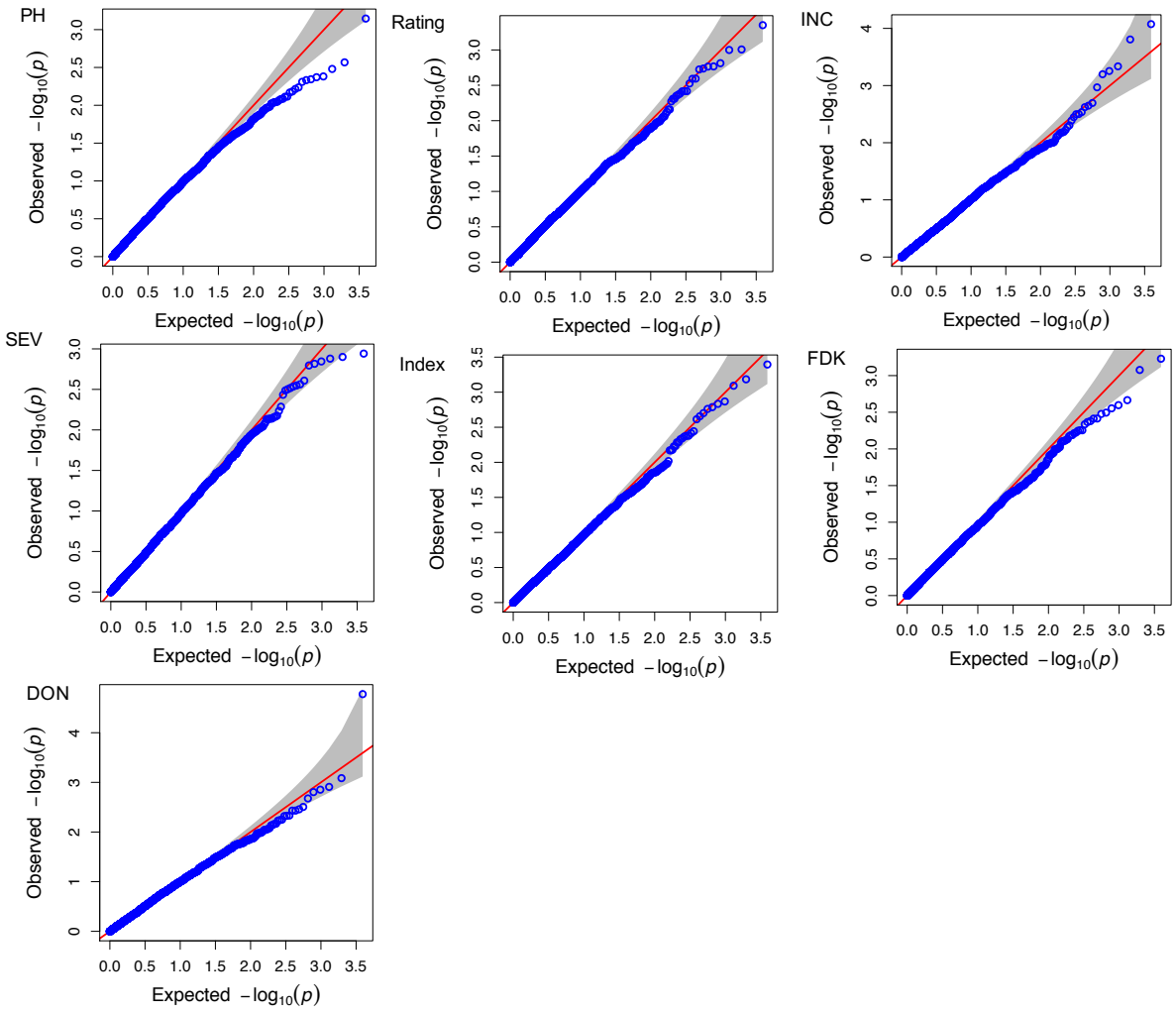
[1]*Ppd-A1a*, *Ppd-B1a* and *Ppd-D1a*, photoperiod insensitive; [2]*Ppd-A1b*, *Ppd-B1b* and *Ppd-D1b,* photoperiod sensitive.



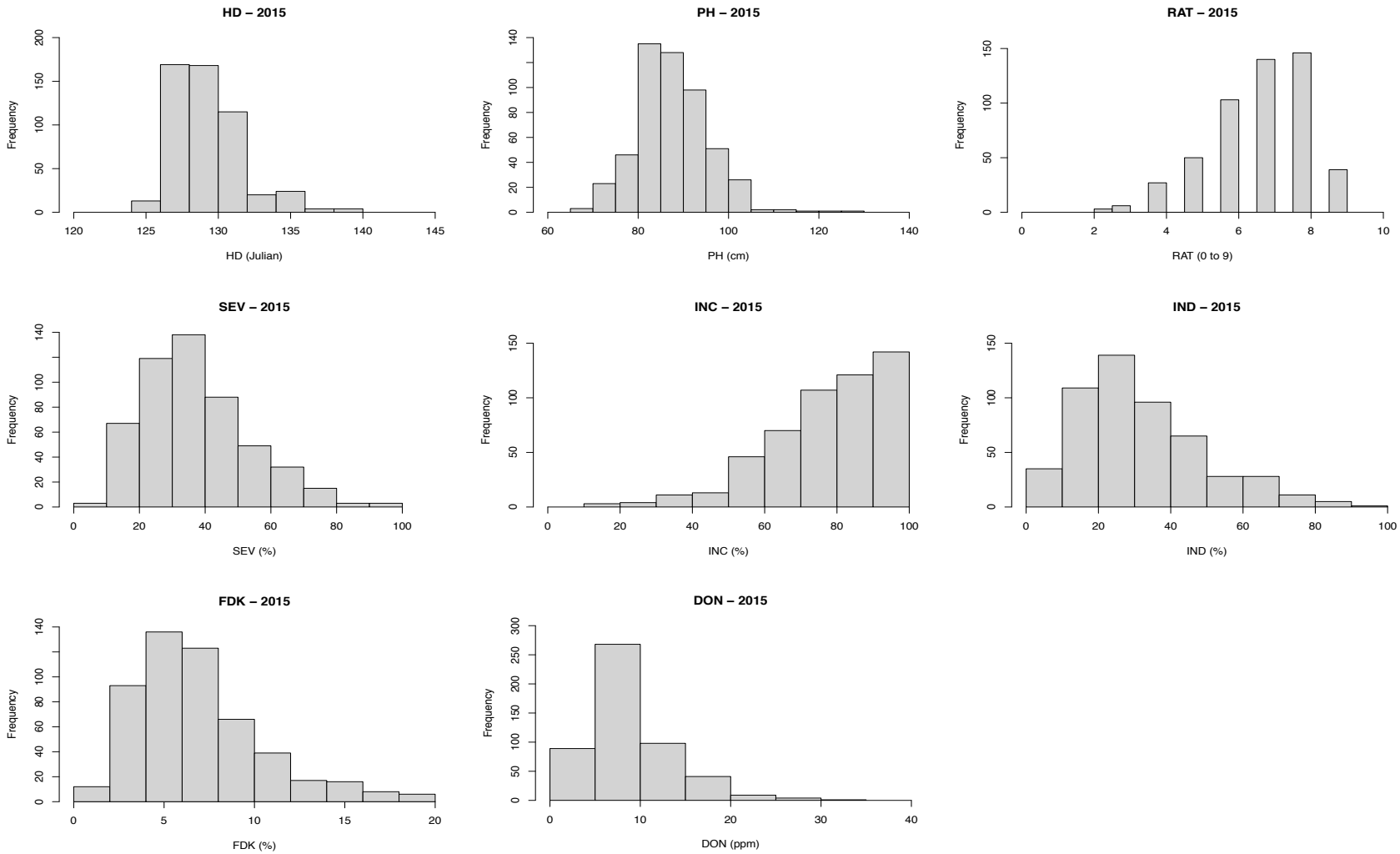
Supplemental Figure S3.1. QQ plots for plant height (PH), FHB rating (Rating), severity (SEV), incidence (INC), Index, Fusarium damaged kernel (FDK) and deoxynivalenol (DON). Analysis using 2015 data of 250 soft red winter wheat cultivars and breeding lines from T-CAP panel grown in Lexington, KY.



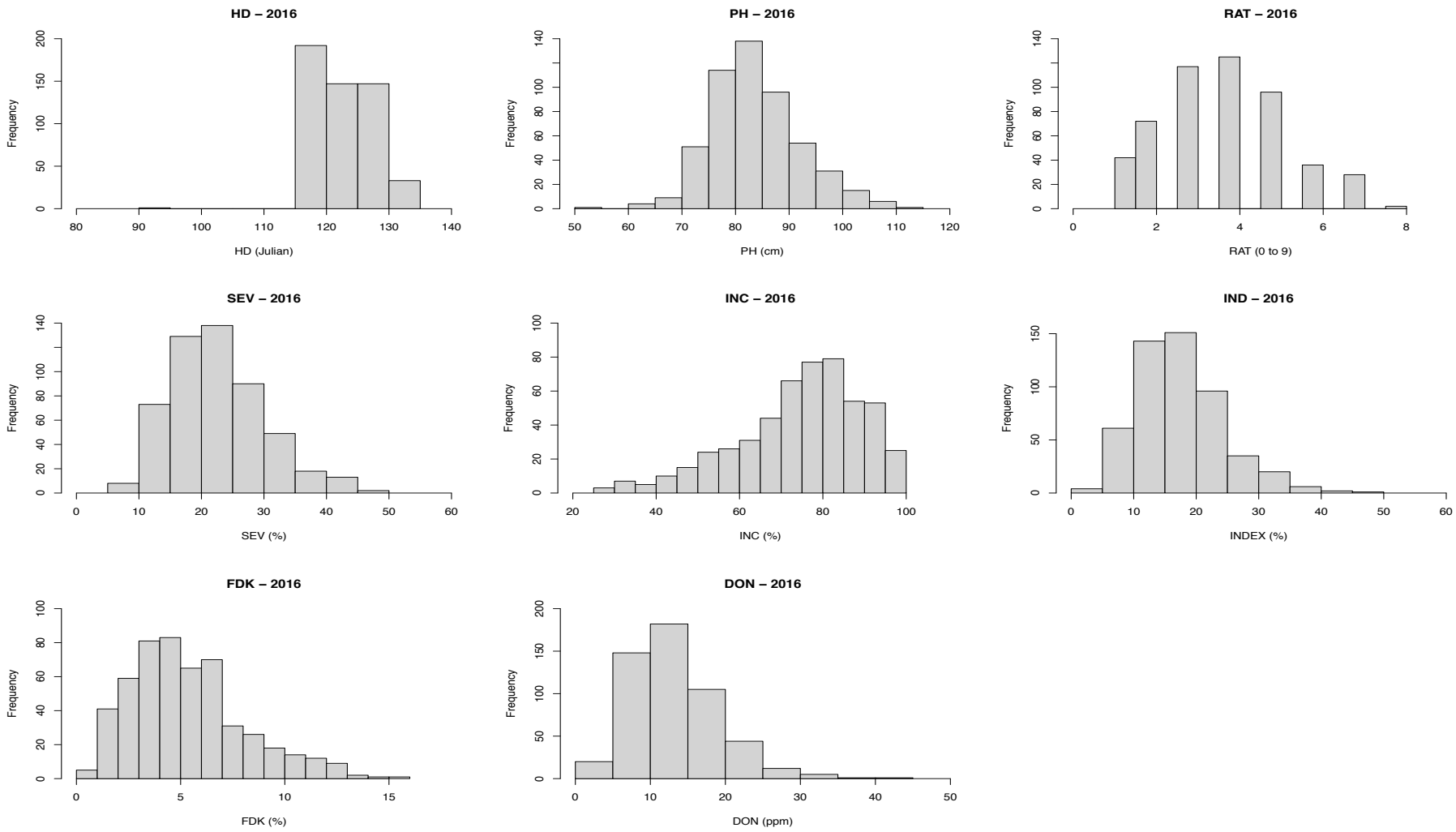
Supplemental Figure S3.2. QQ plots for plant height (PH), FHB rating (Rating), severity (SEV), incidence (INC), Index, Fusarium damaged kernel (FDK) and deoxynivalenol (DON). Analysis using 2016 data of 250 soft red winter wheat cultivars and breeding lines from T-CAP panel grown in Lexington, KY.



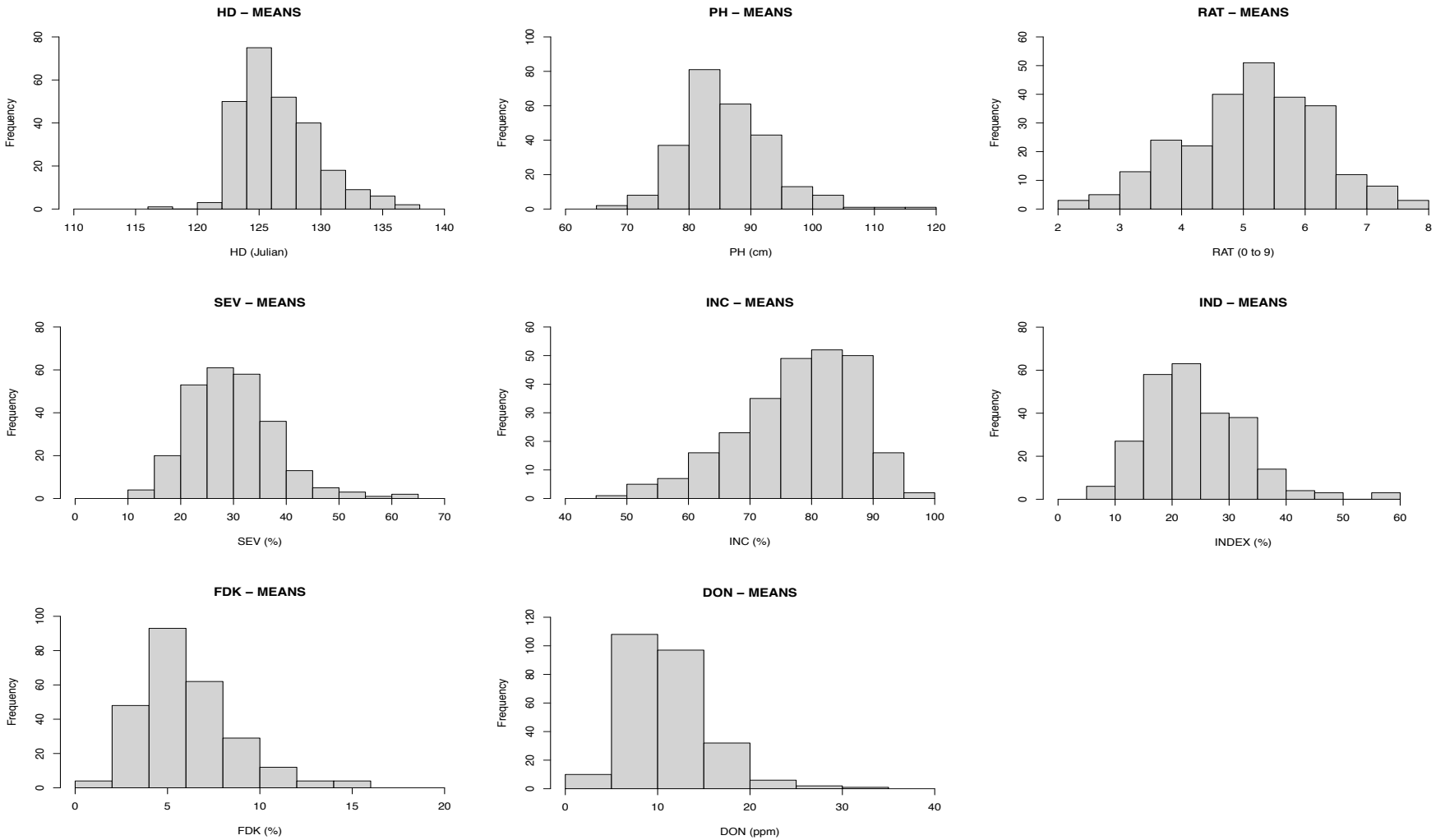
Supplemental Figure S3.3. QQ plots for plant height (PH), FHB rating (Rating), severity (SEV), incidence (INC), Index, Fusarium damaged kernel (FDK) and deoxynivalenol (DON). Analysis using average data (2015 and 2016) of 250 soft red winter wheat cultivars and breeding lines from T-CAP panel grown in Lexington, KY.



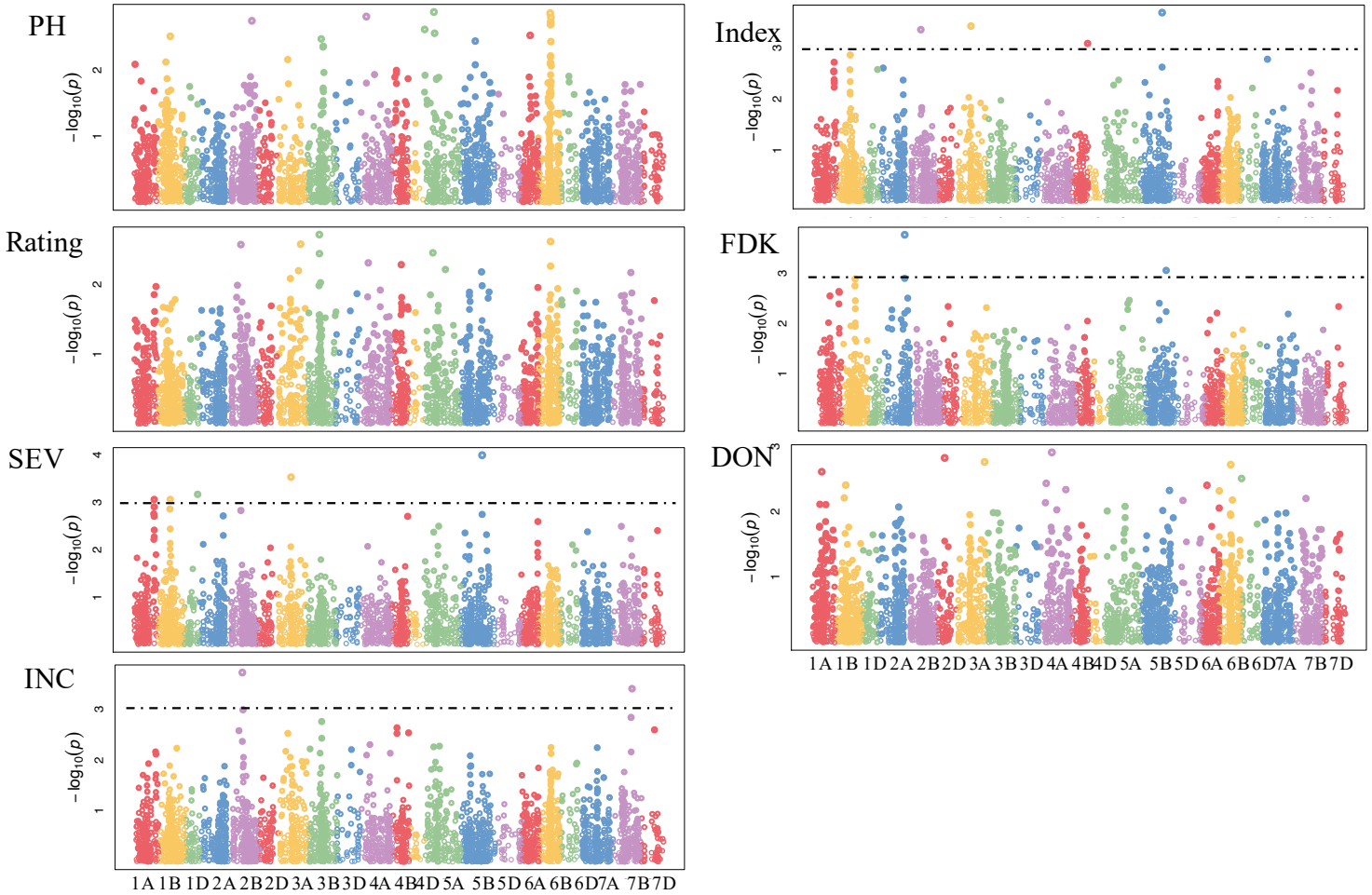
Supplemental Figure S3.4. Histogram for 256 soft red winter wheat cultivars and breeding lines from the T-CAP panel grown in 2014-2015, Lexington, KY.



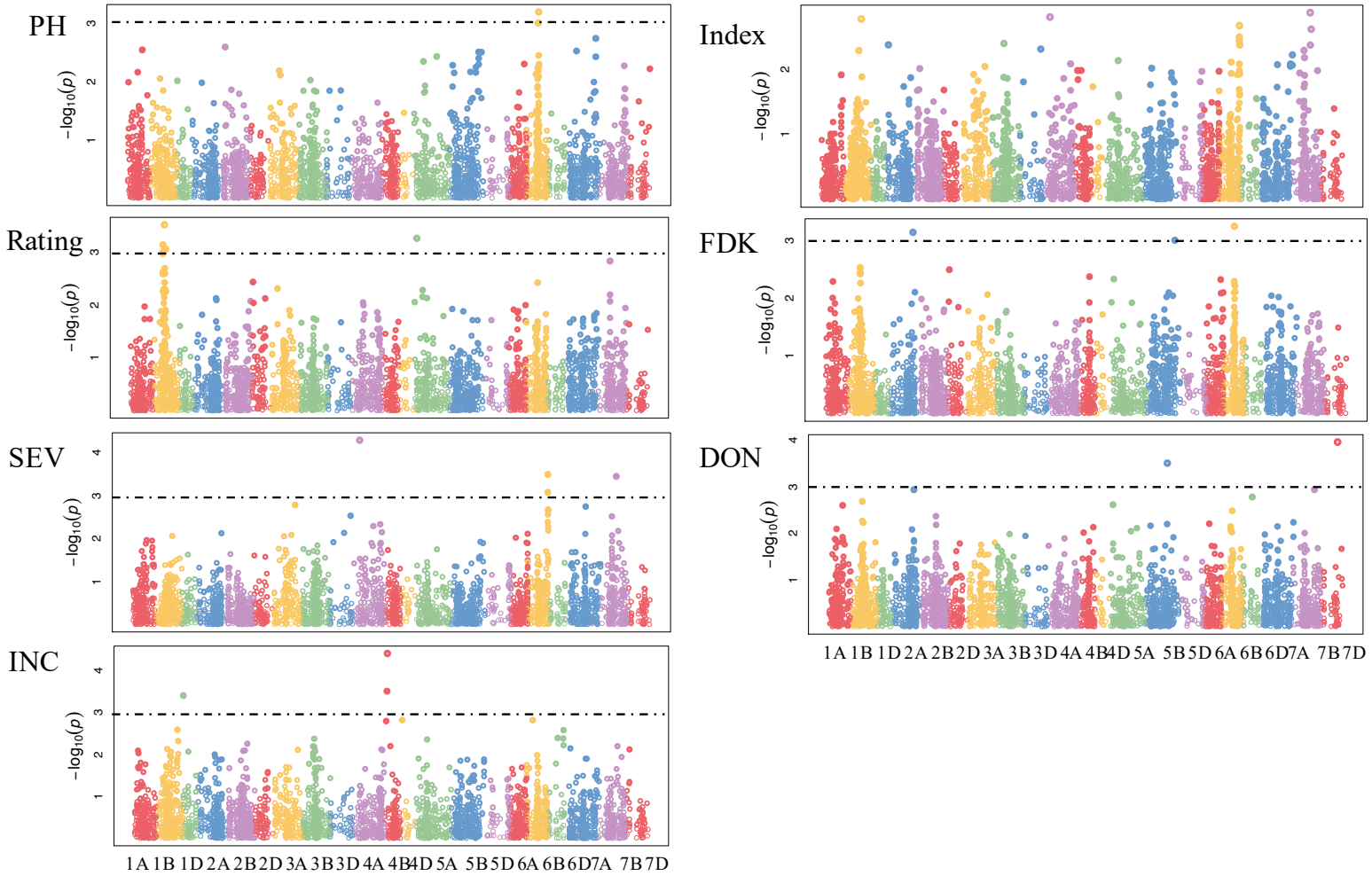
Supplemental Figure S3.5. Histogram for 256 soft red winter wheat cultivars and breeding lines from the T-CAP panel grown in 2015-2016, Lexington, KY.



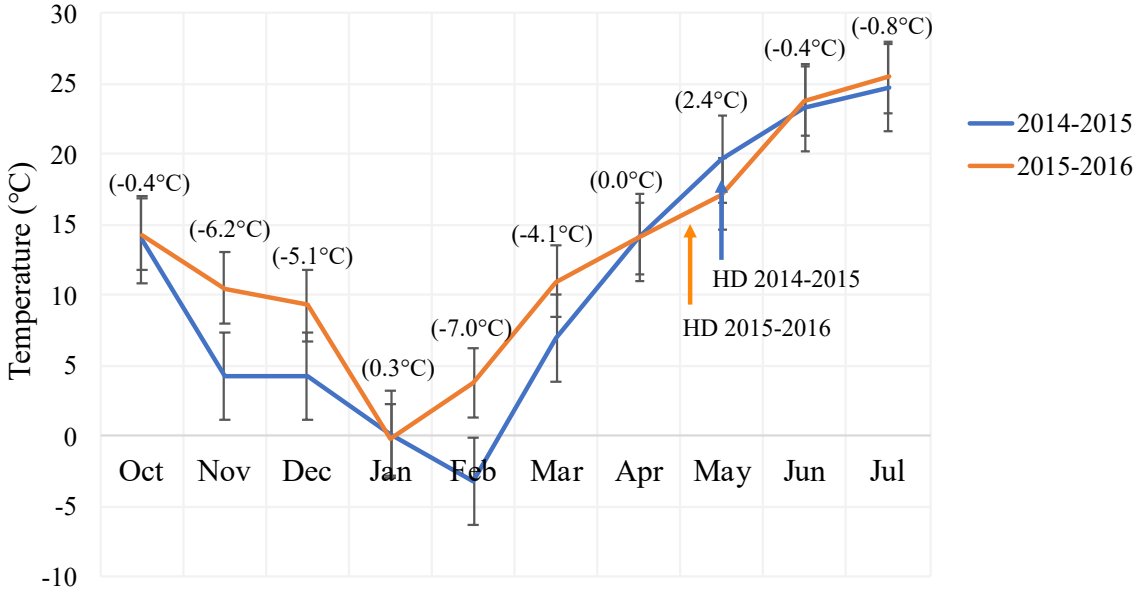
Supplemental Figure S3.6. Histogram for 256 soft red winter wheat cultivars and breeding lines from the T-CAP panel using average data, Lexington, KY.



Supplemental Figure S3.7. Manhattan plots of genome-wide association study (GWAS) was performed for plant height (PH), FHB rating, severity (SEV), incidence (INC), Index, Fusarium damaged kernel (FDK), deoxynivalenol (DON). GWAS results of 250 soft red winter wheat cultivars and breeding lines from T-CAP panel grown in 2014-2015, Lexington, KY.



Supplemental Figure S3.8. Manhattan plots of genome-wide association study (GWAS) was performed for plant height (PH), FHB rating, severity (SEV), incidence (INC), Index, Fusarium damaged kernel (FDK), deoxynivalenol (DON). GWAS results of 250 soft red winter wheat cultivars and breeding lines from T-CAP panel grown in 2015-2016, Lexington, KY.



Supplemental Figure S3.9. Average temperature for the growing seasons of 2014-2015 and 2015-2016, in Lexington, KY. Numbers in the brackets are the difference between the growing season of 2014-2015 and 2015-2016 for each month in Celsius.

ASSOCIATIONS BETWEEN MORPHOLOGICAL AND FHB TRAITS IN A SOFT RED WINTER WHEAT POPULATION

Elisane W. Tessmann, David A. Van Sanford*

Department of Plant and Soil Science, University of Kentucky, Lexington, KY 40546, USA;

*Correspondence: dvs@uky.edu; Tel.:+1-859-338-2409

Received: 28 February 2019, Euphytica

ABSTRACT

Fusarium head blight (FHB) is a destructive disease in wheat (*Triticum aestivum*) and other small grains. Morphological traits can act as barrier between pathogen and plant and provide a passive form of resistance to disease. The objectives were: (i) to phenotype 250 soft red winter wheat (SRWW) breeding lines and cultivars for morphological traits in 2015-16; (ii) to estimate their correlations with disease resistance; and (iii) to identify QTL for morphological traits associated with FHB resistance through a genome-wide association study (GWAS). Morphological traits measured were anther extrusion, plant height, peduncle length, spike length, spikelet number, spike density and spike inclination. Agronomic traits included heading date and yield. Disease traits were: FHB-rating, incidence, severity, FHB-index, Fusarium damaged kernels and deoxynivalenol (DON) concentration. There were significant (p<0.01) differences among genotypes for all morphological traits measured. Moderate heritabilities (0.41-0.66) for spike traits were estimated. Morphological and disease traits were generally negatively correlated; e.g. there was a significant (p<0.01) negative correlation between spike inclination and DON. GWAS identified 29 significant (p<0.001) SNPs associated with morphological traits, ranging from -3.54 to 9.58% of the trait mean. Potential SNPs for yield were located on chromosomes 1B, 3B and 6A. Despite the small effects identified for the SNPs, several morphological traits should be considered for phenotypic and/or genomic selection in FHB resistance breeding programs.

Keywords: morphological traits, Fusarium head blight, GWAS, soft red winter wheat, QTL

Introduction

Fusarium head blight (FHB), is one of the most important diseases in wheat (*Triticum aestivum* L.) and other small grains. Yield reduction, low test weight, reduced percentage of high and low molecular weight glutenins and mycotoxin contamination are some of the effects of this disease (Spanic et al., 2017). FHB is caused by *Fusarium graminearum* a pathogen that likes warm and humid conditions; in cooler, humid environments *F. culmorum* and *F. avenaceum* also can cause the disease (He et al., 2016a). Fungal infection occurs during or just after anthesis and is highly influenced by environmental conditions: warm temperatures in a humid environment with a susceptible host are the triggers for a severe epidemic (Bai and Shaner, 2004; Emrich et al., 2008; Jin et al., 2013).

Modeling distribution of *F. graminearum* showed that this pathogen has been recorded on all continents, except Antarctica, and the authors predict it to be distributed in most of the major rainfed wheat growing regions (Backhouse, 2014). The Intergovernmental Panel on Climate Change (IPCC) released a special issue where they point out that an increase in average temperature of more than 1°C was reached in 2017, and some regions experienced temperature increases of over 1.5°C (Allen et al., 2018). As described in the IPCC report and other studies, increased temperature can change rainfall pattern with some regions having shortage or excess of water which will consequently affect disease development and intensity (Tataw et al., 2016; Allen et al., 2018). In Scotland, for example, FHB is predicted to decrease due to dry conditions during flowering (Skelsey and Newton, 2015). Meanwhile, in northwestern Europe, deoxynivalenol (DON) contamination is predicted to increase up to 3-fold (Fels-Klerx et al., 2012). A recent study

testing the effects of warm rhizosphere showed that FHB levels in wheat were higher in the warming treatment compared to naturally-occurring temperatures (Tessmann and Van Sanford, 2018).

Plant morphology can play an important role during infection and can provide a passive way of resistance or susceptibility to FHB (Steiner et al., 2017; Jones et al., 2018). Morphological traits can act as barriers, reducing the likelihood of contact between pathogen and plant tissue, even if both are present in the environment (Jones et al., 2018). Traits such as plant height, flowering time, anther extrusion, spike density, and spikelet number are some of the traits described in the literature that can be related to FHB (Liu et al., 2007; Graham and Browne, 2009; Suzuki et al., 2012; Liu et al., 2013; Buerstmayr and Buerstmayr, 2016).

Plant height and heading date are well described in the literature in the context of disease resistance. Three mechanisms are reported to be associated with plant height: disease escape, pleiotropy or tight linkage (He et al., 2016a). Disease escape was demonstrated by Yan et al. (2011) using near-isogenic lines; the authors physically raised dwarf genotypes to match wild type (taller) genotypes. The authors observed that disease differences disappeared, and they attributed the results to physical distance where tall genotypes would be distant from the inoculum source. A pleiotropic effect was demonstrated by Saville et al. (2012) studying the function of DELLA, a protein encoded by *Rht-B1b* and *Rht-D1b*. The authors concluded that accumulation of DELLA by the semi-dwarf (*Rht-B1b*) and the severe dwarf allele (*Rht-B1c*) increased susceptibility to initial infection; however, plants with gain of function of DELLA were more resistant to colonization and DON induced cell death. Several studies demonstrated the effects of *Rht-*

B1b and *Rht-D1b* in susceptibility to FHB (Draeger et al., 2007; Miedaner et al., 2011; Srinivasachary et al., 2008, 2009; Lu et al., 2011; Buerstmayr and Buerstmayr, 2016).

Heading date (HD) is also related to FHB, since the pathogen infects the host during flowering, and early vs. late flowering can influence disease levels. Studies demonstrated a positive correlation between heading date and Fusarium damaged kernel (FDK) and DON levels, indicating that early genotypes are more resistant to FHB (Liu et al., 2013; Petersen et al., 2016). Anther extrusion (AE) is also reported to be associated with FHB. Even though wheat is considered an autogamous crop, significant anther extrusion during anthesis has been observed in some cultivars (Muqaddasi et al., 2017). Several investigators have observed an increase in disease levels when anthers were retained in the florets (Graham and Browne, 2009; Skines et al., 2010; Lu et al., 2013; Buerstmayr and Buerstamayr, 2015). Skines et al. (2010) suggested that lines in which anthers were trapped between glumes were an easy target for colonization by *Fusarium* because they provided dead tissue. In addition, a study demonstrated that plants with closed florets and/or rapid anther extrusion were more resistant to FHB than lines with partially extruded anthers (Kubo et al., 2013).

Spike characteristics such as length, number of florets, density and inclination can be associated with passive disease resistance. Spike density, for instance, can influence the microclimate around florets, such that a dense spike could increase humidity and thus favor fungal development (Jones et al., 2018). Positive correlations between spike density and FHB severity have been reported (Buerstmayr et al., 2011; Giancaspro et al., 2016; Yi et al., 2018). However, a negative correlation between spike density and FHB incidence was observed by Steiner et al. (2004). In barley (*Hordeum vulgare*), Urrea et al. (2002) also

identified a negative association between FHB severity and spike density. For spikelet number, neither Liu et al. (2007) nor Buerstmayr et al. (2011) observed significant correlations with FHB, while Jones et al. (2018) found a positive correlation with DON. These results demonstrated the variability of morphological traits in relation to FHB response. Thus, field experiments with a large and diverse set of lines are fundamental for a proper characterization of the effects of morphological traits in disease response. In this sense, our objective was to analyze 250 soft red winter wheat (SRWW) cultivars and breeding lines for morphological traits and their relationship with disease resistance. Lines used in this study came from breeding programs across the eastern region of the United States. Our main objectives were: (i) to phenotype 250 SRWW cultivars and breeding lines for morphological traits; (ii) to estimate correlations between these morphological traits and disease resistance; and (iii) to identify QTL for morphological traits associated with FHB resistance through a genome wide association study (GWAS).

Materials and Methods

Morphological trait experiment

The study was conducted at the University of Kentucky Spindletop Research Farm in Lexington, KY (38°7′37.81″ N, 84°29′44.85″ W). Soil type at the site is a Bluegrass Maury silt loam (fine, mixed, semiactive, mesic Typic Paleudalfs).

We planted 250 SRWW cultivars and breeding lines from the Elite Eastern Mapping Panel of the Triticeae Coordinated Agricultural Project (TCAP;

http://www.triticeaecap.org/). The TCAP project involved 21 states and 55 Universities across the US, and was funded by the USDA - National Institute for Food and Agriculture.

Our experiment was conducted during the growing seasons of 2014-2015 and 2015-2016, hereafter referred to by their respective harvest years, 2015 and 2016. The planting dates were 23 October 2014 and 19 October 2015. The experimental design consisted of a completely randomized design, where each plot had six rows 1.2 m long, spaced 0.3 m between rows.

To evaluate important morphological traits, ten random plants per plot were marked with tape in the peduncle before any measurements were made. The following traits were evaluated on the marked plants: anther extrusion (AE), plant height (PH, cm), peduncle length (PL, cm), spike length (SL, cm), spikelet number (SN), spike density (SD), and spike inclination (SI).

Anther extrusion was a visual estimation of how much the anther had emerged from the floret. We evaluated this trait three to four days after the main spike was completely out of the flag leaf sheath. To avoid overestimation of AE, plots were checked daily for flowering since variation in temperature can influence flowering. Underestimation due to wind removal of the anthers was avoided by opening florets and checking for the presence of anthers. We used a scale from 0 (0 – 25%) to 3 (75 – 100%) for extruded anthers. High temperatures during the flowering period in 2014-15 shortened the window for flowering and reduced the magnitude of variation among genotypes. For this reason, we decided not to evaluate AE in 2016.

Selected tillers were harvested by hand at the soil level, and morphological traits were measured in laboratory. Plant height, peduncle length, spike length, spikelet number,

spike density, and spike inclination were evaluated for each cultivar and breeding line. Plant height was the distance from soil surface to the top of spike, excluding awns. Peduncle length was measured as the distance from the upper node to the basal node of the spike. Spike length was the distance from the basal node of the spike to the top of the spike, excluding awns. Spikelet number was counted as the total number of fertile spikelets per spike. Spike density was calculated as the spikelet number divided by spike length. Spike inclination was a visual estimation ranging from 1 (vertical spike, equivalent to a 90° angle) to 4 (more than a 180° angle, parallel to the soil surface). The plot area was equivalent to 2.33 m^2. Plots were harvested with a research combine, and yield was assessed for each plot.

Scab nursery experiment

To evaluate passive resistance conferred by morphological traits, we conducted a second experiment to quantify FHB resistance in the mapping panel. More information about the experimental design is available in Tessmann et al. (chapter 3). The following traits were evaluated: heading date (HD), FHB incidence (INC), FHB severity (SEV), FHB index, Fusarium damaged kernels (FDK), and deoxynivalenol concentrations in harvested grain (DON).

Heading date was recorded when more than 50% of the spikes in the row had emerged (in Julian days). Around 24 days after HD, INC, SEV and FHB rating were evaluated. Incidence was assessed by counting the number of blighted spikes in a random sample of 20 spikes (in percentage). To arrive at an estimate of severity, we counted the number of infected spikelets per total number of spikelets in 10 blighted heads (expressed

as percentage). FHB index was obtained by multiplying severity and incidence, and multiplying the product by 100 (expressed as percentage). FHB rating was a visual estimate of FHB index ranging from 0 (absence of FHB symptoms) to 9 (≥ 90% of FHB blighted spikelets).

Lines were manually harvested using a sickle, mechanically threshed and cleaned. After cleaning, a sample of 15 g from each row was further cleaned by hand and evaluated for FDK. An air separation machine specifically developed from a Precision Machine head thresher and a Shop-Vac vacuum was used to separate scabby kernels from healthy ones for FDK as described in Agostinelli et al. (2012). Scabby and healthy kernels were weighed separately and FDK was calculated by:

$$FDK\ (\%) = \frac{W_{sk}}{W_{sk} + W_{hk}}\ x\ 100$$

where W_{sk} = weight scabby kernel (g); and W_{hk} = weight healthy kernel (g). The same sample (15 g) was sent to the University of Minnesota DON testing laboratory for DON analysis. DON concentration was determined by gas chromatography with mass spectrometry (Mirocha et al., 1998; Fuentes et al., 2005).

Genome wide association study

The 250 SRWW cultivars and breeding lines were used in a genome wide association study. All entries in the mapping panel were genotyped with the 90 K Illumina SNP chip to identify single nucleotide polymorphisms (SNP). The USDA-ARS Biosciences Research Laboratory, Fargo, ND, US, carried out the genotyping process. The initial number of markers was approximately 28000; however, after removing markers with minor allele frequency < 10%, missing data > 5% and using SNP tagging, the final number

of independent markers was 3919 (Mao, personal communication, 2017). The process of removing markers was conducted by the lab group of Dr. Clay Sneller at The Ohio State University (Columbus, OH). Our study used the 3919 markers for the GWAS, using the genomic association and prediction integrated tool (GAPIT; Lipka et al. 2012). GAPIT uses a compressed mixed linear model approach to identify SNP associated with the traits of interest. The model used can be expressed as follows:

$$Y = X\beta + Zu + e$$

In which Y = represents the phenotype; β = is an unknown fixed effect vector that contains genetic marker, population structure and intercept; u = unknown vector of random additive genetic effects from multiple background QTL for individuals or lines; X and Z = are known matrices; e = residual. We used the following QTL as covariates in the model: *Rht-B1*, *Rht-D1*, *Vrn-A1*, *Vrn-B1*, *Vrn-D3*, *Ppd-A1*, *Ppd-B1* and *Ppd-D1*. Population structure was analyzed using TASSEL (http://www.maizegenetics.net) software, and there was no underlying structure identified in the analysis.

SNPs associated with yield identified through GAPIT were then used to assess yield, FHB rating, FDK and DON levels in the lines containing each allelic form for the SNPs as follows: First, we classified the lines for genotype at the specific SNP; next, disease levels were assessed for each of these lines, and mean comparisons using a t-test was performed between lines with the contrasting alleles at the SNP.

Statistical analysis

Analysis of variance (ANOVA) was performed using the General Linear Models procedure in SAS (Proc GLM; SAS Institute Inc. Version 9.3) to determine the significance of the main effect genotype (cultivar and breeding lines). The model used was:

$$Y_{ij} = \mu + G_i + Y_j + G_i * Y_j + \varepsilon_{ij}$$

Where: Y_{ij} = the observation of the *ith* genotype in the *jth* year; μ = the overall mean; G_i = the effect of *ith* genotype; Y_j the effect of *jth* year; G_i * Y_j = the effect of the interaction of the *ith* genotype and the *jth* year, ε_{ij} = the residual error.

Broad sense heritability of the traits measured in this study was estimated on an entry mean basis using the model above. The expected mean squares (EMS) and heritability were obtained by using Proc Varcomp in SAS (SAS Institute Inc., Version 9.3). The following equation was used to estimate heritability:

$$h^2 = \frac{\sigma_g^2}{\sigma_p^2}$$

Where: h^2 = heritability, σ_g^2 = genotypic variance, σ_p^2 = phenotypic variance. Confidence intervals (90%) were calculated after Knapp et al. (1985) as:

$$UL = 1 - \left[\left(\frac{MS3}{MS2}\right) x\ FUL(0.10, v1\ and\ v2\ df)\right]^{-1}$$

$$LL = 1 - \left[\left(\frac{MS3}{MS2}\right) x\ FLL(0.90, v1\ and\ v2\ df)\right]^{-1}$$

Where: UL and LL= upper and lower limit of the confidence interval, respectively, MS3 = entry mean square, MS2 = residual mean square, FUL and FLL = F value for the upper and lower limits, respectively.

Anther extrusion was only measured in 2014-2015; thus, the broad sense heritability estimate was termed "repeatability" (Holland and Nvquist, 2003) and the model used was:

$$Y_j = \mu + G_i + \varepsilon_i$$

where effects are defined as above.

Correlations among traits were estimated using entry means in JMP (SAS Institute Inc. Version 13.2).

Results

Phenotypic analysis

To assess variability among the genotypes in this study, we performed mean comparisons and analysis of variance for eight morphological traits (Table 4.1). Genotype effects were significant for all traits evaluated in this study as was the year effect (p<0.01). The results for year and genotype x year interaction are shown only for traits evaluated in both years (Table 4.1).

We observed a significant (p<0.05) increase in the mean value of PH, PL, SL and SD observed in 2016 over that measured in 2015 (Table 4.1). Increases of 5.9 and 8.8% were observed for PH and PL for example, respectively. We saw the same trend for the spike traits SL and SD where increases of 4.0 and 1.9% were recorded, respectively. No differences between years were detected for spikelet number. For SI and yield we observed a significant (p<0.05) decrease in the overall means for 2016 compared to the 2015 means. Spike inclination decreased by 31.6%, meaning that spikes were more erect in 2016 than

in 2015. Similarly, yield decreased 24.6% in 2016. Anther extrusion was only evaluated in 2015 after we decided that our environmental conditions, with high temperatures during flowering, made it difficult to evaluate the trait accurately. However, the evaluation in 2015 gave us an idea of how the population in this study expressed this trait, with 50-75%, on average, of anther extrusion for the genotypes.

Heritability estimates are also presented in Table 4.1. Repeatability of anther extrusion was 0.93. Heritability of plant height was moderately high (0.77), in agreement with previous studies in our group (Balut et al., 2013; Tessmann and Van Sanford, 2018). Peduncle length also had high heritability (0.74). Moderate heritability was also observed for spike length, spikelet number, spike density and spike inclination.

Correlations among morphological traits evaluated in 2015 and 2016 are shown in Table 4.2. Significant correlation values ranged from -0.56 (p<0.01; SD with SN, 2016) to 0.70 (p<0.01; PH with PL, in 2015). Plant height was positively correlated with all traits in both years, with the exception of SD and AE in 2015 and SD in 2016. For instance, plant height and peduncle length had a positive correlation of 0.70 (p<0.01) in 2015 and 0.58 (p<0.01) in 2016.

Spike length had strong positive correlations with spikelet number and spike density in both years and with spike inclination and yield only in 2016. Spikelet number had a strong negative correlation with spike density in both years. In addition, spikelet number was positively correlated with spike inclination (p<0.01) and negatively correlated with yield (p<0.05) in 2015; while in 2016, spikelet number was positively correlated with yield (p<0.01). Spike density was negatively correlated with spike inclination in 2015.

Anther extrusion was negatively correlated with peduncle length and spike density and positively correlated with spikelet number.

Yield was positively correlated in 2015 with plant height, peduncle length, spikelet number and spike inclination and positively correlated in 2016 with plant height, spike length and spikelet number (Table 4.2). Anther extrusion was negatively correlated with plant height and spike density, and positively correlated with spikelet number.

Correlations between morphological and FHB traits are shown in Table 4.3. Plant height was negatively correlated with all FHB traits evaluated and positively correlated with heading date. A similar trend was observed for peduncle length which had negative correlations with all FHB traits, and a positive correlation with heading date.

For traits related to spike morphology, we observed a positive correlation of heading date with spike length and spikelet number. Spike length was negatively correlated with all FHB traits except DON. Values of -0.32 (p<0.01) and -0.14 (p<0.05) were observed for the correlations of SL and FHB rating and SL and FDK, respectively. Spikelet number also was negatively correlated with all traits, except FHB incidence and DON, and we can highlight the correlation between SN and FHB rating with -0.32 (p<0.01). Spike density was only correlated with INC, where a negative relationship was observed between traits. Spike inclination, like other spike traits, was negatively correlated with FHB traits. In other words, a spike more parallel to the soil surface would provide more resistance to FHB than an erect spike. Spike inclination was the only spike trait correlated with DON, with value of -0.17 (p<0.01).

Yield was positively correlated with heading date (0.13; p<0.05), which was negatively correlated with all FHB traits, with the exception of incidence, where no

correlation was observed (Table 4.3). Anther extrusion was not correlated with any of the FHB traits measured in this study.

Genome wide association study (GWAS)

We performed GWAS to identify promising QTLs associated with morphological traits that can confer resistance to FHB. In the analysis we also used important growth and development QTLs such as *Rht-B1*, *Rht-D1*, *Vrn-A1*, *Vrn-B1*, *Vrn-D3*, *Ppd-A1*, *Ppd-B1*, and *Ppd-D1* as covariates in the model. The GWAS was conducted using 2-year entry means for all traits with the exception of AE; only SNPs with a LOD score > 3 were considered. Manhattan plots for AE, PH, PL, SL, SN, SD, SI and yield are presented in Figure 4.1. A total of 29 SNPs were detected across this study. SNPs were located on chromosomes 1B, 2A, 2B, 2D, 3A, 3B, 3D, 4B, 5B, 6A, 6B, 7A and 7B (Figure 4.1, Table 4.4). Potential SNPs and the magnitude of their effects are presented in Table 4.4. Effects for the SNPs ranged from -3.54 to 9.58%.

Association mapping identified very small effect, less than 1%, for all traits with the exception of yield (Table 4.4). For instance, AE had an association on chromosome 4B with a rather small effect (0.07%). Three SNPs were detected in the analysis for PH where two SNPs had positive effect (chr. 1B and 3A), increasing PH, and one SNP had a negative effect, reducing plant height (chr. 3D). Four SNPs were identified for peduncle length: two SNPs, M13089 and M5021, reduced peduncle length by respective magnitudes of -0.17 and -0.40%. The other two SNPs, M1657 and M4594, had the reverse effect.

GWAS for spike traits identified 6, 1, 3 and 5 SNPs for SL, SN, SD and SI, respectively. Chromosomes 2B, 3A, 5B and 6B were identified for SL with effects ranging

from -0.02 to 0.02%. Spike density SNPs were identified on chromosomes 2D and 7A. SNPs for SI were identified on chromosomes 2D, 3B, 5B and 7B. The association mapping for yield identified 6 SNPs (Figure 4.1, Table 4.4). Negative effects were observed for the SNPs M1696 (chr. 1B) and M9370 (chr. 5B) while SNP M1641 on chromosome 1B had an effect of 2.35% on yield. Similarly, SNP M6375, on chromosome 3B, had positive effect of 4.84% on yield. The last two SNPs, on chromosomes 5B and 6A, had positive effects of 8.96 and 9.58% on yield, respectively.

In order to assess the effects of the SNPs identified through the GWAS associated with yield, we classified the entries based on their genotype at each SNP. In addition to yield, we also look at the effects of these SNPs on FHB rating, FDK and DON due to their negative correlation with yield. After classifying the lines for the alternative allele at the SNP, we used phenotypic data to assess yield and disease levels (Table 4.5). For SNP M1696, a decrease in yield of 13.2% was associated the TT genotype. Disease levels for this SNP increased FDK and DON by 26.3 and 14.0%, respectively.

SNP M1641, on chromosome 1B, was associated with a yield increase of 8.4% and reductions of 13.7 and 27.6% in FHB rating and FDK, respectively, when the genotype TT was in the population. Similarly, M6375 was implicated in a yield decrease of 5.1% when TT was in the population; however, no differences were observed between the contrasting genotypes of the SNP for the disease traits. TT alleles at SNP M9363 on chromosome 5B was associated with decreased yield (7.6%). The opposite scenario was observed when the TT genotype of the SNP M10354 was present; we observed a yield increase of 6.3%. The last SNP detected in the GWAS was M9370; we observed a yield reduction of 7.9% and increased DON (11.6%) associated with the TT genotype.

Due to the importance of the *Rht*, *Vrn* and *Ppd* loci on growth and development in wheat, we looked at the effects of each allelic form of the QTL for the traits evaluated in this study (Table 4.6). Alternative alleles at these QTL did not have a significant effect on peduncle length, spike length and yield. Plant height, on the other hand was increased 3.2% when the *Ppd-D1b* allele was in the population: 79.4 vs. 81.9 cm for *Ppd-D1-insensitive* and *sensitive* alleles, respectively. Significant differences in spikelet number was observed between the alleles of *Ppd-B1* and *Ppd-D1*: we found increases of 7.5 and 3.3% associated with the *Ppd-B1b* and *Ppd-D1b* alleles, respectively, when they were present (Table 4.6). For the last two traits, spike density and inclination, significant differences were only observed between the allelic forms of *Ppd-D1*: a decrease of 3.9% for spike density and an increase of 14.3% for spike inclination were associated with the *Ppd-D1b* allele.

Discussion

Morphological traits

Morphological traits can confer passive resistance to FHB through plant characteristics that potentially reduce contact between plant and pathogen. The resistance to initial infection, in this case, is not conferred by disease resistance genes per se (Jones et al., 2018). Thus, field evaluations for morphological traits can deliver insights about the plant-pathogen relationship. Significant differences among genotypes were observed for all traits evaluated in both years of this study (Table 4.1). These results indicate that there is genetic diversity for morphological traits in the diverse TCAP mapping panel. An overall look at trait means each year showed that in 2016 plants were taller, with longer peduncles,

and spikes were more compact and erect than in 2015, and with an average yield 24.6% lower than in 2015 (Table 4.1).

An analysis of the water supply during the growing seasons of 2014-2015 and 2015-2016 showed a drastic difference in precipitation pattern between years (Figure S4.1). An increase of 49.5 and 80.3 mm during the vegetative stages in November and December, respectively, was observed in 2016 when compared with 2015. In addition, temperature was higher for the same period; 6.2 and 5.1°C for November and December, respectively (Figure S4.2). In March and April, when we have the transition from vegetative to reproductive growth, a considerable reduction in precipitation was observed in 2016 when compared with the previous year; 53.9 and 117.9 mm, respectively (Figure S4.1). In June, during the grain filling period, high temperatures and low water availability were observed in 2016 when compared with 2015 (Figure S4.1 and S4.2). Overall temperatures were higher during the entire growing season of 2016 and water availability was variable, with lower amounts during important growth stages which could potentially have affected plant development and yield.

The association between grain number and yield have been reported in many studies (Sinclair and Jamieson, 2006; Garcia et al., 2014; Quintero et al., 2018). In our study, there were no differences for average spikelet number in either year; however, there were differences among mapping panel entries (Table 4.1). For example, in 2015 spikelet number ranged from 11.7 to 20.8, while in 2016 it ranged from 12.3 to 19.5 (data not presented). A small negative correlation was observed between SN and yield ($p<0.05$) in 2015, while in 2016 the correlation was positive ($p<0.01$; Table 4.2). Despite the correlations, there were no significant differences for SN, on average, between years, thus

we cannot explain yield reduction in 2016 by changes in spikelet number. Sinclair and Jamieson (2006) mentioned a study conducted by Gooding et al. (2003), where the authors demonstrated that drought stress after anthesis did not change grain number, but it did decrease grain weight. This trade-off could be due to a source limitation during grain filling (Sinclair and Jamieson, 2006). Quintero et al. (2018) demonstrated that grain number and weight in wheat depends on the effect of the environment.

Yield was negatively correlated with all FHB traits, with the exception of incidence (Table 4.3). These results are not surprising since this association is well described in the literature (Nganje et al., 2002; Johnson et al., 2003; Reis and Carmona, 2013; Salgado et al., 2015).

The relationship between morphological and disease traits is presented in Table 4.3. Overall, we observed negative correlations among all traits, with the exception of heading date, where a positive correlation was observed with PH, PL, SL, SN and yield. Heading date is a major adaptive trait and can help maximize yield potential in different environments (Snape et al., 2001; Kiseleva et al., 2016). In this study, the positive correlation of HD and the other traits, suggested that late flowering genotypes were higher yielding and taller, with elongated peduncles and spikes with higher spikelet number (Table 4.3). Some studies suggest a pleiotropic effect of anthesis on post-anthesis leaf senescence, grain yield and grain protein concentrations (Bogard et al., 2011; Zanke et al., 2014). Plant height was negatively correlated with all disease traits, which agrees with the literature (Hilton et al., 1999; Klahr et al., 2007; Srinivasachary et al., 2008, 2009; Mao et al., 2010; Yan et al., 2011; Kollers et al., 2013; Lu et al., 2011, 2013; Buerstmayr and Buerstmayr, 2016; Schulthess et al., 2018).

The semi-dwarfing alleles *Rht-B1b* and *Rht-D1b* are widely used in breeding programs, though these two semi-dwarfing genes express different levels of susceptibility to disease (Steiner et al., 2017). Srinivasachary et al. (2009) demonstrated an increased susceptibility to initial infection (type I) when *Rht-B1b* and *Rht-D1b* were present in the genotype. They also found that *Rht-B1b* increased resistance to spread of the pathogen within the spike (type II). Other studies also reported *Rht-D1b* to be associated with decrease in resistance to initial infection (Draeger et al., 2007; Srinivasachary et al., 2008; Lu et al., 2011). In this study, the QTL analyses showed that the different alleles at the *Rht* loci did not significantly affect plant height (Table 4.6). However, when looking at disease traits such as DON, FDK and FHB rating with respect to the *Rht* loci we observed that plants with *Rht-D1b* allele were more susceptible to FHB with higher levels of DON and FHB rating than plants with *Rht-B1b* allele (Figure 4.2). Thus, our results are in accordance with the previously cited literature.

Besides heading date and plant height, the major focus of the study was on those morphological traits that reduce the likelihood of contact between susceptible tissue and pathogen (Jones et al., 2018). A negative correlation was observed between spike length and all disease traits, with exception of DON (Table 4.3). In other words, in our study, plants with shorter spikes had higher levels of FHB. Similar results were found by Buerstmayr et al. (2011) studying a population from the cross between *Triticum macha x T. aestivum*, where a negative correlation was observed between FHB severity and spike length. Similarly, Suzuki et al. (2012) found a negative correlation between FDK and spike length. In the present study, spikelet number was negatively correlated with FHB rating, severity, FHB index and FDK (Table 4.3). Liu et al. (2007) and Buerstmayr et al. (2011)

found no significant correlation between spikelet number and FHB, while Jones et al. (2018) observed a positive correlation. Jones et al. (2018) suggested that the relationship of spike traits and FHB could be dependent on the dispersal mechanism of the pathogen and interactions with the environment. The authors pointed out that due to the different mechanisms by which *Fusarium* is spread, spike traits could play an important role in disease occurrence and intensity. It is interesting that the QTL analysis for spikelet number showed differences associated with *Ppd-B1* and *Ppd-D1* sensitive and insensitive alleles (Table 4.6). An increase of 7.3% in spikelet number was observed in *Ppd-B1* sensitive genotypes when compared with those carrying the insensitive allele. Similar, a spikelet number increase of 3.3% was observed for *Ppd-D1* sensitive allele.

Spike density (SD) can affect the growing environment for the pathogen because its architecture impacts the humidity within the spike microclimate (Jones et al., 2018). Thus, a lax spike would experience increased air movement between spikelets which could affect negatively fungal growth. In our study, we did not identify significant correlations of SD with the traits evaluated, with the exception of a small negative correlation with incidence. Similar results were found by Steiner et al. (2004), where a small negative correlation between spike density and incidence and spread was observed in the cultivar Frontana. The authors suggested that several small effect loci controlled spike density and unlinked genes were responsible for FHB resistance and spike density. In contrast with these results, Buerstmayr et al. (2011) and Giancaspro et al. (2016) reported a positive correlation between spike density and severity although the two traits were not correlated in the present study (Table 4.3). Our QTL analysis showed a small (-3.9%) but significant

(p<0.05) difference between *Ppd-D1* sensitive and insensitive alleles in their effect on spike density (Table 4.6).

Another morphological trait evaluated was spike inclination (SI). The relationship of SI and FHB relies on the hypothesis that upright spikes can hold water for a longer period of time which could favor disease development since it provides a humid environment for the pathogen (Mamo and Steffenson, 2015). In this study, spike inclination was negatively correlated (p<0.01) with all disease traits indicating that more inclined spikes had lower disease levels (Table 4.3). For FDK and DON, correlations with spike inclination were -0.23 and -0.17, respectively. Similar results were observed in barley where a negative correlation was obtained for spike angle and FHB severity (Urrea et al., 2002). The QTL analysis for spike inclination showed an increase of 14.3% in inclination when the *Ppd-D1* sensitive allele was present in the genotypes (Table 4.6).

GWAS

A challenge for this century is to increase production of cultivars resistant to biotic and abiotic stress without increasing the land area under production. Advances in genomics with reduction in costs for genome sequencing are making it possible to explore genetic diversity in different populations. In this sense, genomic selection is a tool that takes advantage of phenotypic and genotypic data and can deliver information about the areas of the genome involved in the traits studied. Here, we evaluated 250 soft red winter wheat cultivars and breeding lines from the Eastern mapping panel. Eight morphological traits were evaluated over two years in Lexington, KY, and GWAS identified 29 SNPs for these

traits (Figure 4.1). SNP effects ranged from -3.54 to 9.58% and chromosomes 1B, 2A, 2B, 2D, 3A, 3B, 3D, 4B, 5B, 6A, 6B, 7A and 7B were identified in the analysis (Table 4.4).

Very small effects were observed for all morphological traits (Table 4.4), ranging from -0.0001 (SD) to 0.55% (PH). Despite the small effects observed from the GWAS analysis, these traits were negatively correlated with all FHB traits evaluated in this study (Table 4.3). In addition, high heritability estimates were observed for all traits, with the exception of spike inclination that had moderate heritability (Table 4.1). Thus, phenotypic evaluation and selection for these morphological traits, in conjunction with genomic selection could improve resistance to FHB.

The GWAS analysis for yield identified 6 SNPs with effects ranging from -3.54 to 9.58% (Table 4.4). Two SNPs had a negative effect on yield, while the other 4 SNPs, M1641, M6375, M9363 and M10354, had positive effects on yield and could potentially be used in breeding programs (Table 4.4).

SNPs M1641, M6375 and M10354 with the genotype TT were associated with yield increases of 8.4, 5.1 and 6.3%, respectively (Table 4.5). SNPs M1696 and M9370 increased FDK and DON levels while reducing yield when the genotype TT was present (Table 4.5). SNP M1641 reduced FHB rating by 13.7 %, FDK by 27.6% and increased yield by 8.4%. This SNP could potentially be used in breeding, but it would have to be validated in various genetic backgrounds and replicated, multi-environment tests. Similarly, the SNPs on chromosomes 3B and 6A also could be valuable in breeding programs, given the same caveats. Turuspekov et al. (2017), suggested that the environment conditions where the genotypes are grown can affect the identification of QTLs for yield due to its genotype x environment interaction. It is important to note that

yield was measured in single plots without replications in the current study, thus its estimation is not as reliable as estimates based on conventional replicated yield trials (Hall and Van Sanford, 2003).

GWAS can help illuminate the genetic architecture of a trait, providing information of the number of genes controlling a specific trait and their potential effects (Schimid and Bennewitz, 2017). Huang and Han (2014), suggested using the information from GWAS for candidate gene identification and using targeting induced local lesions in genomes (TILLING) for gene validation. SNPs identified in the GWAS can also be used in genomic selection (GS), where highly significant SNPs would be used as fixed effects in the GS model (Begum et al., 2015). In our study, the morphological traits had very small SNP effects, below 1% (-0.0001 to 0.55%), though there were SNPs with larger effects associated with yield. In this sense, a candidate gene approach would not be indicated for those traits. The SNPs identified for yield could be potentially used when devising crosses to increase yield and disease resistance. Based on SNP effects in the population for yield, we can also suggest the SNPs M1641 (chr.1B), M6375 (chr.3B) and M10354 (chr.6A) for potential use in genomic selection due to their effects estimated in the GWAS.

Conclusion

This study demonstrates the importance of morphological traits in disease resistance. Negative correlations between morphological and scab traits were observed across all traits in this study. Despite their small effects, traits such as spike length, spikelet number and spike inclination should be considered when phenotyping the population. Due

to the negative correlation with FHB traits, these spike traits can function as passive resistance mechanisms and potentially reduce pathogen contact with the floral tissue. The GWAS for morphological traits identified a total of 29 SNPs with very small effect QTLs for all traits, except yield. Three potential SNPs with positive effects were identified on chromosomes 1B (M1641), 3B (M6375) and 6A (M10354) for yield though these effects require validation in other genetic backgrounds and in multi-environment replicated tests.

Acknowledgments

This work was founded by grants from USDA Triticeae Coordinate Agricultural Project, N 59-0206-4-002 and the U.S. Department of Agriculture, through the US Wheat and Barley Scab Initiative under Agreement No. 59-0206-9-054. We thank Anthony Clark, John Connelley and Sandy Swanson for technical assistance.

Compliance with ethical standards

Conflict of Interest

The authors declare that they have no conflict of interest.

Table 4.1. Means of 250 soft red winter wheat lines for morphological traits evaluated in Lexington, KY, 2015-2016. Below the means, mean squares and level of significance for genotype, year, and genotype x year (G x Y) are shown for each trait evaluated. Broad sense heritability estimates for all traits, except AE (repeatability values) and 90% confidence interval (lower limit (LL) and upper limit (UL)), 2015 and 2016 are shown in the bottom panel.

	PH[1]	PL[2]	SL[3]	SN[4]	SD[5]	SI[6]	AE[7]	Yield[8]
				Means				
2015	78.30 B	30.87 B	8.06 B	15.57 A	0.52 B	1.77 A	2.16	924.50 A
2016	82.90 A	33.59 A	8.38 A	15.80 A	0.53 A	1.21 B	.	697.32 B
				ANOVA				
Genotype	1053.78**	271.86**	9.16**	35.52**	0.03**	2.85**	4.63**	23059.30*
Year	26349.84**	9522.57**	126.68**	68.25**	0.19**	412.07**	.	6708988.90**
G x Y	238.15**	69.62**	3.08**	15.47**	0.01**	1.68**	.	.
CV[9]	5.35	9.49	9.67	9.94	8.55	38.18	25.92	16.41
				Broad sense Heritability				
h^2	0.77	0.74	0.66	0.57	0.62	0.41	0.93†	.
LL	0.72	0.69	0.59	0.47	0.53	0.52	0.92	.
UL	0.82	0.79	0.73	0.65	0.69	0.28	0.94	.

[1]PH, plant height (cm); [2]PL, peduncle length (cm); [3]SL, spike length (cm); [4]SN, Spikelet number; [5]SD, spike density; [6]SI, spike inclination (0 to 4); [7]AE, anther extrusion (0 to 3); [8]yield (g).

† Repeatability value

[9]CV= coefficient of variation, ** $p \leq 0.01$, $^{*}p \leq 0.05$

Table 4.2. Correlations among morphological traits evaluated in a study conducted in Lexington, KY, 2015-2016. Above diagonal are the correlations for 2015, and below diagonal are the correlations for 2016.

		2015							
		PH[1]	PL[2]	SL[3]	SN[4]	SD[5]	SI[6]	Yield[7]	AE[8]
2016	PH	.	0.70**	0.27**	0.31**	-0.09ns	0.27**	0.40**	-0.10ns
	PL	0.58**	.	0.20**	0.05ns	0.19**	0.31**	0.27**	-0.24**
	SL	0.50**	0.32**	.	0.71**	0.33**	0.06ns	-0.11ns	-0.04ns
	SN	0.39**	0.18**	0.55**	.	-0.42**	0.18**	-0.13*	0.18**
	SD	0.06ns	0.10ns	0.38**	-0.56**	.	-0.17**	0.02ns	-0.29**
	SI	0.20**	0.03ns	0.16**	0.08ns	0.06ns	.	0.13*	0.04ns
	Yield	0.32**	0.07ns	0.23**	0.23**	-0.03ns	0.07ns	.	-0.07ns
	AE	.	.	.	.	.	.	.	.

[1]PH, plant height (cm); [2]PL, peduncle length (cm); [3]SL, spike length (cm); [4]SN, Spikelet number; [5]SD, spike density; [6]SI, spike inclination (0 to 4); [7]yield (g); [8]AE, anther extrusion (0 to 3).

** $p \leq 0.01$, * $p \leq 0.05$, ns not significant at *t* test

Table 4.3. Pearson correlations based on 2 year entry means among morphological and scab disease traits in a diverse wheat mapping panel evaluated in 2015 and 2016, Lexington, KY.

		Scab traits						
		HD[9]	FHB Rating[10]	SEV[11]	INC[12]	FHB Index[13]	FDK[14]	DON[15]
Morphological traits	PH[1]	0.61**	-0.51**	-0.32**	-0.27**	-0.34**	-0.46**	-0.25**
	PL[2]	0.25**	-0.41**	-0.35**	-0.30**	-0.37**	-0.35**	-0.26**
	SL[3]	0.45**	-0.32**	-0.18**	-0.19**	-0.20**	-0.14*	0.03^{ns}
	SN[4]	0.50**	-0.32**	-0.17**	-0.05^{ns}	-0.14*	-0.15*	0.01^{ns}
	SD[5]	-0.06^{ns}	0.02^{ns}	-0.01^{ns}	-0.16**	-0.05^{ns}	0.03^{ns}	0.05^{ns}
	SI[6]	-0.02^{ns}	-0.24**	-0.22**	-0.18**	-0.23**	-0.23**	-0.17**
	Yield[7]	0.13*	-0.33**	-0.34**	-0.05^{ns}	-0.30**	-0.42**	-0.38**
	AE[8]	0.11^{ns}	0.03^{ns}	0.08^{ns}	0.05^{ns}	0.09^{ns}	-0.01^{ns}	-0.10^{ns}

[1]PH, plant height (cm); [2]PL, peduncle length (cm); [3]SL, spike length (cm); [4]SN, Spikelet number; [5]SD, spike density; [6]SI, spike inclination (0 to 4); [7]yield (g), [8]AE, anther extrusion (0 to 3); [9]HD, heading date (Julian date); [10]FHB, Rating Fusarium head blight rating (0 to 9); [11]SEV, severity (%); [12]INC, incidence (%); [13]FHB, index (%); [14]FDK, Fusarium damaged kernels (%); [15]DON, deoxynivalenol (ppm).

** $p \leq 0.01$, $^{*}p \leq 0.05$, ^{ns} not significant at *t* test

Table 4.4. GWAS of 250 soft red winter wheat lines grown in 2015-2016, Lexington, KY. Only SNPs with LOD score > 3 are shown. Effect of SNPs is expressed in percentage of the mean of each trait.

Trait	SNP	Chr.[1]	cM[2]	*p value*	Effect (%)	R^2(w/o SNP)	R^2(w/SNP)
AE[3]	M7550	4B	211.01	*0.00030*	0.07	0.03213	0.08607
PH[4]	M6687	3D	280.71	*0.00036*	-0.28	0.07652	0.12677
	M1657	1B	269.73	*0.00062*	0.55	0.07652	0.12265
	M5010	3A	218.69	*0.00084*	0.09	0.07652	0.12030
PL[5]	M1657	1B	269.73	*0.00042*	0.01	0.02616	0.07791
	M4594	2D	142.23	*0.00073*	0.47	0.02616	0.07351
	M13089	7B	497.57	*0.00074*	-0.17	0.02616	0.07340
	M5021	3A	242.85	*0.00094*	-0.40	0.02616	0.07150
SL[6]	M4328	2B	458.55	*0.00028*	0.02	0.09026	0.14166
	M4361	2B	489.33	*0.00034*	0.02	0.09026	0.14017
	M11418	6B	375.21	*0.00043*	-0.02	0.09026	0.13831
	M8584	5B	62.86	*0.00047*	0.001	0.09026	0.13766
	M9325	5B	397.52	*0.00067*	-0.01	0.09026	0.13507
	M5506	3A	380.63	*0.00091*	0.002	0.09026	0.13283
SN[7]	M2959	2A	467.84	*0.00041*	-0.04	0.08046	0.12951
SD[8]	M4468	2D	25.49	*0.00019*	-0.001	0.06700	0.12249
	M12411	7A	635.34	*0.00038*	0.003	0.06700	0.11734
	M12417	7A	642.19	*0.00041*	-0.0001	0.06700	0.11674
SI[9]	M4448	2D	8.15	*0.00056*	0.03	0.03451	0.08354
	M9038	5B	213.22	*0.00056*	-0.02	0.03451	0.08352
	M12963	7B	384.54	*0.00073*	0.01	0.03451	0.08138
	M5748	3B	226.99	*0.00085*	0.02	0.03451	0.08021
	M9116	5B	222.57	*0.00087*	-0.03	0.03451	0.08006
Yield[10]	M1696	1B	287.98	*0.00026*	-3.38	0.03782	0.09284
	M1641	1B	269.73	*0.00030*	2.35	0.03782	0.09169
	M6375	3B	320.47	*0.00038*	4.84	0.03782	0.08973
	M9363	5B	412.97	*0.00058*	8.96	0.03782	0.08638
	M10354	6A	271.03	*0.00067*	9.58	0.03782	0.08524
	M9370	5B	416.45	*0.00085*	-3.54	0.03782	0.08343

[1]Chr., Chromosome; [2]cM, centimorgan; [3]AE, anther extrusion (0 to 3); [4]PH, plant height (cm), [5]PL, peduncle length (cm); [6]SL, spike length (cm); [7]SN, spikelet number; [8]SD, spike density; [9]SI, spike inclination (0 to 4); [10]yield (g).

Table 4.5. Effects of six SNPs on yield, FHB rating, Fusarium damaged kernels (FDK) and deoxynivalenol (DON) for 250 soft red winter wheat lines grown in 2015 and 2016, Lexington, KY. Number of lines of each allelic class shown in parentheses.

SNP		Yield	FHB Rating	FDK	DON
M1696 (Chr.1B)	TT (27)	724.6 B	5.6 A	8.0 A	12.9 A
	AA (193)	820.0 A	5.2 A	5.9 B	11.1 B
M1641 (Chr.1B)	TT (191)	824.3 A	5.1 B	5.8 B	10.8 A
	AA (37)	755.5 B	5.8 A	7.4 A	12.4 A
M6375 (Chr.3B)	TT (95)	836.5 A	5.3 A	5.9 A	10.8 A
	AA (153)	793.8 B	5.2 A	6.2 A	11.1 A
M9363 (Chr.5B)	TT (189)	796.0 B	5.3 A	6.1 A	10.9 A
	AA (58)	856.2 A	5.3 A	6.1 A	11.4 A
M10354 (Chr.6A)	TT (139)	831.3 A	5.3 A	6.0 A	10.8 A
	AA (101)	779.0 B	5.3 A	6.2 A	11.5 A
M9370 (Chr.5B)	TT (63)	765.5 B	5.4 A	6.6 A	12.1 A
	AA (183)	825.8 A	5.3 A	6.0 A	10.7 B

Within columns, means followed by the same letter are not significantly different according to *t* test (0.05)

Table 4.6. Quantitative trait locus (QTL) effect on plant height (PH), peduncle length (PL), spike length (SL), spikelet number (SN), spike density (SD), spike inclination (SI), and yield means of 250 soft red winter wheat lines grown in 2015 and 2016 in Lexington, KY.

QTL	Number of lines	PH	PL	SL	SN	SD	SI	Yield
Rht-B1a	120	80.9 A	31.9 A	8.2 A	15.7 A	0.53 A	1.5 A	807.4 A
Rht-B1b	126	80.4 A	32.4 A	8.2 A	15.6 A	0.53 A	1.5 A	811.0 A
Rht-D1a	157	80.4 A	32.4 A	8.2 A	15.7 A	0.53 A	1.5 A	814.2 A
Rht-D1b	89	80.6 A	31.6 A	8.2 A	15.6 A	0.53 A	1.5 A	801.2 A
Vrn-A1	236	80.7 A	32.2 A	8.2 A	15.6 A	0.53 A	1.5 A	811.5 A
Vrn-A1-short	14	79.8 A	31.5 A	8.3 A	16.0 A	0.53 A	1.4 A	788.3 A
Vrn-B1	242	80.7 A	32.2 A	8.2 A	15.7 A	0.53 A	1.5 A	810.8 A
Vrn-B1-short	5	78.1 A	33.4 A	7.8 A	15.2 A	0.52 A	1.8 A	813.9 A
Vrn-D3b	171	80.6 A	32.1 A	8.2 A	15.6 A	0.53 A	1.5 A	807.3 A
Vrn-D3b-early	76	80.5 A	32.4 A	8.3 A	15.8 A	0.53 A	1.5 A	815.9 A
Ppd-A1a	140	80.3 A	32.4 A	8.2 A	15.6 A	0.53 A	1.5 A	822.9 A
Ppd-A1b	104	81.1 A	31.9 A	8.3 A	15.8 A	0.53 A	1.4 A	799.6 A
Ppd-B1a	19	78.5 A	32.1 A	7.9 A	14.7 B	0.54 A	1.4 A	807.3 A
Ppd-B1b	189	80.9 A	32.1 A	8.2 A	15.8 A	0.53 A	1.5 A	809.1 A
Ppd-D1a	123	79.4 B	32.2 A	8.2 A	15.4 B	0.54 A	1.4 B	806.3 A
Ppd-D1b	123	81.9 A	32.2 A	8.2 A	15.9 A	0.52 B	1.6 A	812.5 A

Rht-B1a and *Rht-D1a,* height wild type allele; *Rht-B1b* and *Rht-D1b,* dwarfing height allele; *Vrn-A1*, *Vrn-B1* and *Vrn-D3,* vernalization allele; *Vrn-A1-short*, *Vrn-B1-short* and *Vrn-D3-early,* without vernalization allele; *Ppd-A1a*, *Ppd-B1a* and *Ppd-D1a,* photoperiod insensitive; *Ppd-A1b*, *Ppd-B1b* and *Ppd-D1b,* photoperiod sensitive.

Within columns, means followed by the same letter are not significantly different according to *t* test (0.05)

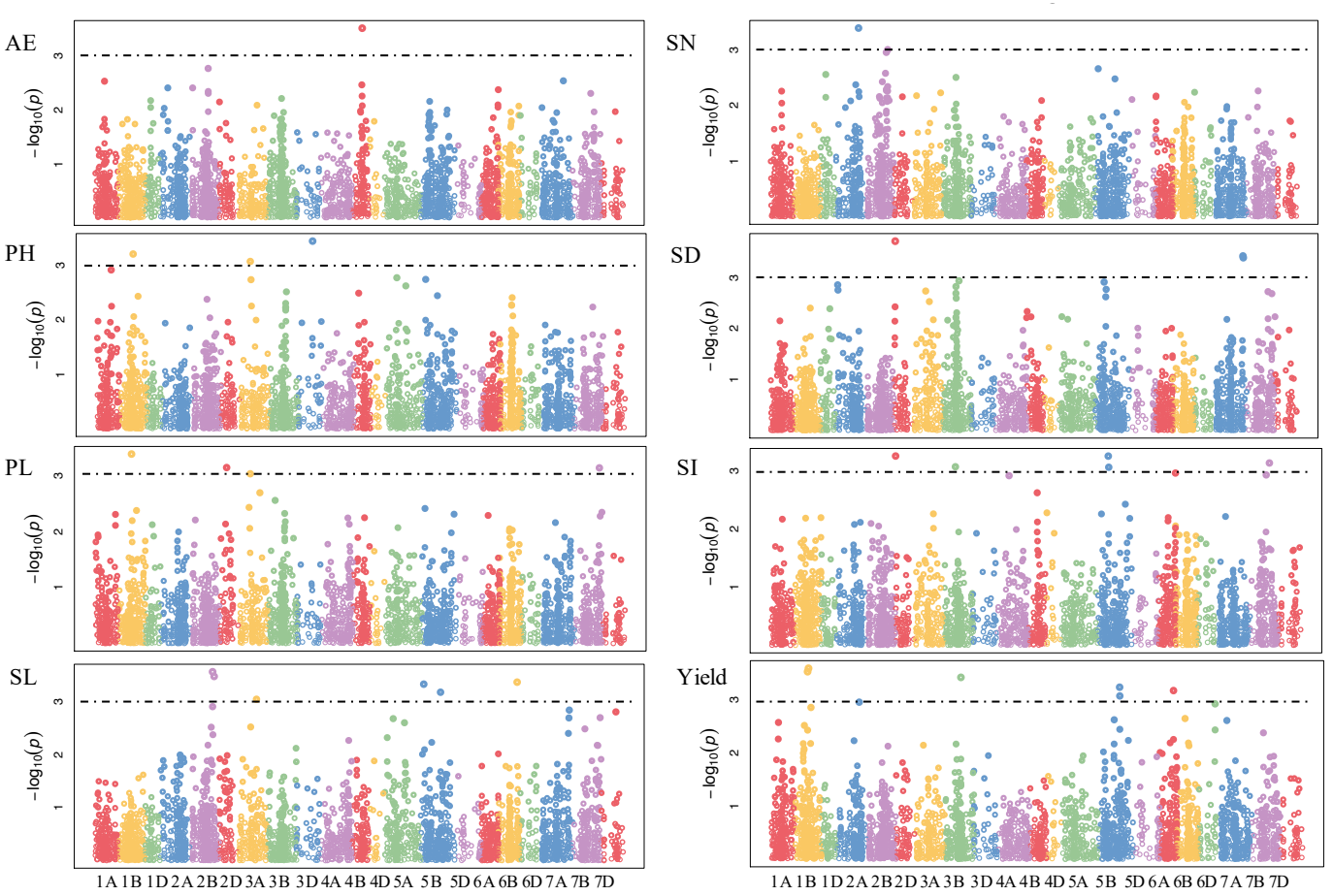


Figure 4.1. Manhattan plots from the genome-wide association study (GWAS) that was performed for anther extrusion (AE), plant height (PH), peduncle length (PL), spike length (SL), spikelet number (SN), spike density (SD), spike inclination (SI), and yield. The GWAS was conducted on 250 soft red winter wheat cultivars and breeding lines from T-CAP wheat mapping panel grown in 2015 and 2016, Lexington, KY.

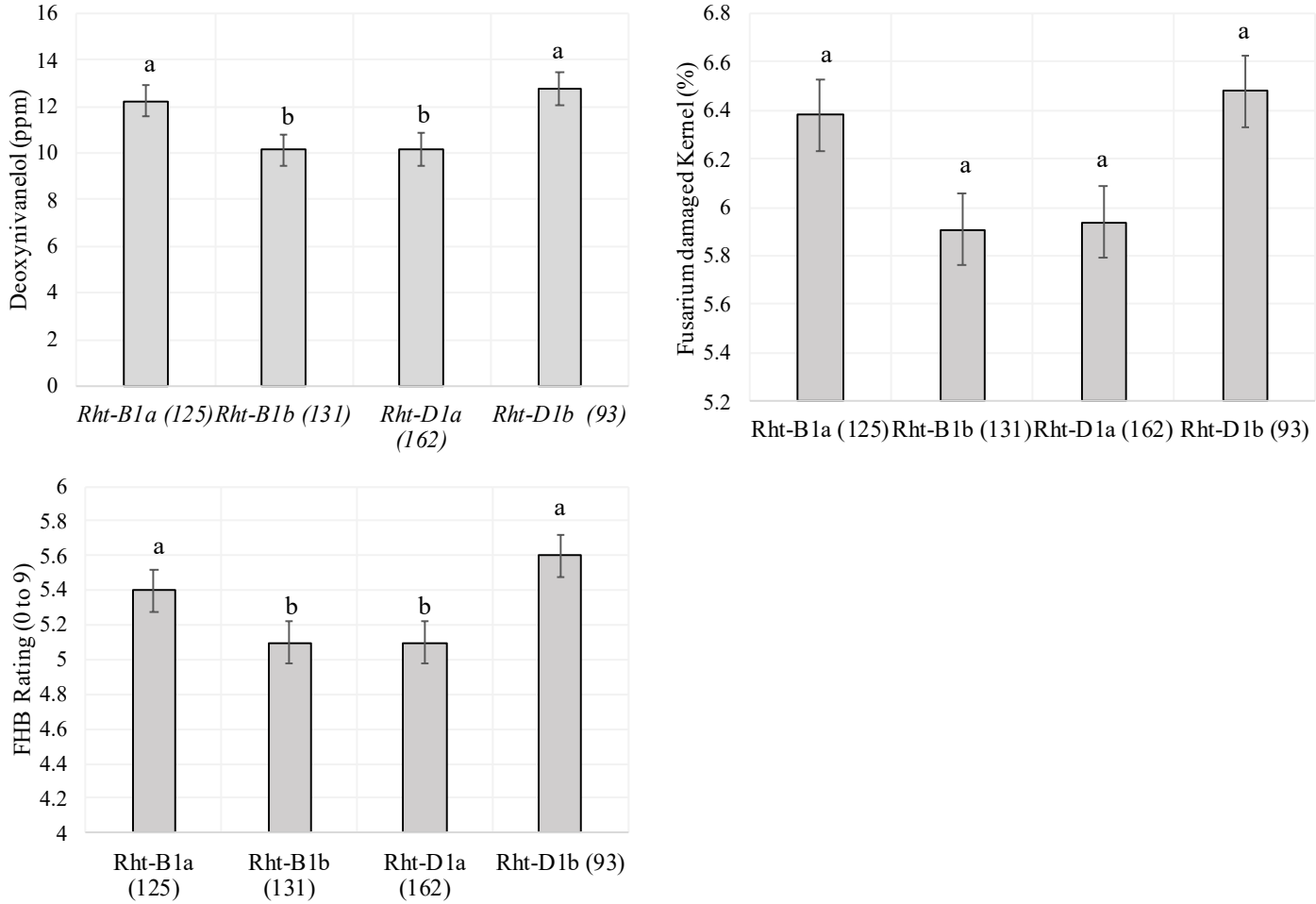
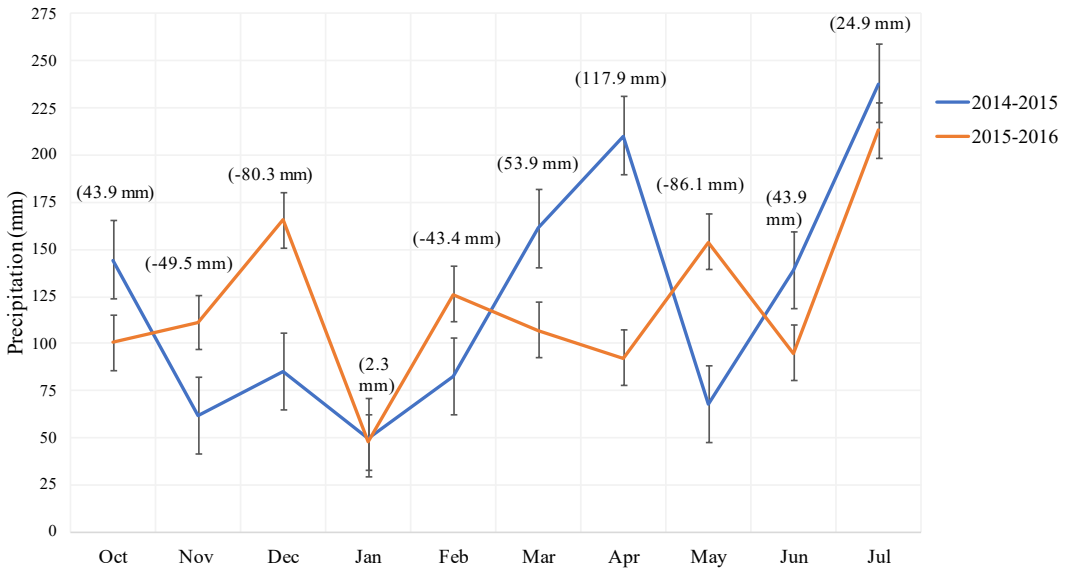
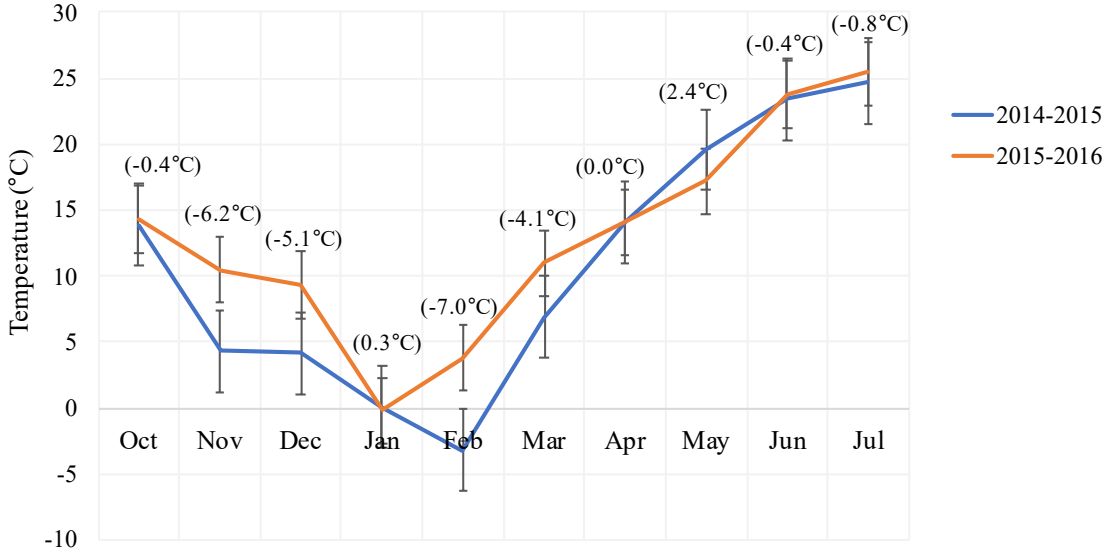


Figure 4.2. Allelic effects of dwarfing genes on deoxynivalenol, Fusarium damaged kernels and FHB rating in 250 wheat lines grown in Lexington, KY, during the growing seasons 2014-2015 and 2015-2016. Values in parentheses represent the number of genotypes that have the allele.



Supplemental Figure S4.1. Average precipitation for the growing season of 2014-2015 and 2015-2016, in Lexington, KY. Number in brackets are the difference in millimeter between the two growing seasons for each month (weather.uky.edu/ky/climate.php).



Supplemental Figure S4.2. Average temperature for the growing seasons of 2014-2015 and 2015-2016, in Lexington, KY. Number in brackets are the difference in degrees Celsius between the two growing seasons for each month (weather.uky.edu/ky/climate.php).

SUMMARY

How to increase yield production in the next decades in an environment that is constantly changing? This is probably one of the most important challenges faced by scientists worldwide. Increases in temperature are already a reality with some regions experiencing increases of up to 1.5 ºC. With these temperature increases come changes in rain distribution which can affect plant diseases occurrence and intensity. In wheat, Fusarium head blight (FHB) is a very important disease that causes yield losses by kernel damage and toxin production, and it is driven by environmental conditions. In this scenario, it is important to understand how climate change can affect FHB level in genotypes used in breeding programs and how morphological traits can provide disease resistance to FHB.

As mention before in this dissertation, the complexity of FHB reflects weather conditions (warm and humid environment), plant growth stage and susceptibility of the host. In this dissertation I have discussed three studies to assess the effects of FHB on a diverse set of breeding lines and cultivars: GWAS for Fusarium head blight related traits in winter wheat (*Triticum aestivum*) in an artificially warmed treatment; GWAS for Fusarium head blight traits in the current natural environment; and association between morphological and FHB traits. My overall goals were to identify the effects of increased temperature on FHB and to identify morphological traits that could provide a passive source of resistance.

In the first study I simulated an increase in temperature by using cables buried at the soil level. An increase of ~ 2ºC in temperature was sufficient to cause a shift of ~3.5 days in heading date in both years of this study. This is important because it could be the

difference in having the disease or not. In fact, for the disease traits I saw a drastic effect of the warmed treatment. For example, DON and FDK, increased 84% and 131% for the genotypes in the warmed treatment, respectively. QTL such as *Fhb1*, height (*Rht*), vernalization (*Vrn*) and photoperiod (*Ppd*) genes, are broadly used in breeding programs. Under warmed conditions the genotypes had higher levels of DON and FDK independent of the allelic form of these QTLs. I looked at the best 15 performers in the population, and we observed, in general, that the superior genotypes in the control treatment were also superior in the warmed treatment. These genotypes have the potential to be used in breeding programs targeting FHB resistance in a warmed environment. However, more field experiments to evaluate genotypes under warmed conditions. In terms of the possible QTL involved, the GWAS identified 19 and 10 SNPs associated in the control and warmed treatments, respectively. SNP effects were very small ranging from -2.5 to 2.6%. Although the SNPs had small effects, SNPs identified for the warmed study can be useful in genomic selection programs.

The second study used the same large and diverse set of lines to identify promising QTL associated with FHB resistance. The goals were to phenotype the population and conduct a GWAS analysis. As expected, there was genetic diversity for all traits evaluated in this population. Disease levels were higher in 2015 than in 2016, and I attributed that to differences in temperature during both growing seasons, where a cooler environment in 2016 during fungal colonization probably slowed down the development of disease symptoms. I looked at lines with the 20 highest and lowest DON concentration levels along with QTL for plant height, vernalization, photoperiod and *Fhb1*. I observed that, on average, DON levels were 5.0 and 21.6 ppm for the low DON and high DON groups,

respectively. These are important numbers, when considering that the FDA guideline is 1 ppm in processed food. The group with lowest DON concentration ranged from 3.7 to 5.7 ppm and those lines were taller, on average, than the group with highest DON concentration and none of those genotypes had the *Fhb1* gene. The GWAS analysis identified 16 SNPs associated with FHB traits and their effects ranged from -2.14 to 4.01% of the mean of a given trait. Negative effect SNPs were associated with FHB and DON on chromosomes 5B, 6B and 4A. A decrease of 2.1, 1.5 and 3.2 ppm in DON levels was observed when the genotype TT was in the population versus genotype AA. These results suggest that even a small effect QTL can potentially reduce DON levels and, thus, be useful in breeding programs.

The third study focused on morphological traits and their potential to provide passive resistance mechanisms to FHB. We know that major and minor genes are involved in conferring resistance to FHB, however, morphological structures can play an important role by acting as barrier between pathogen and plant tissue and thus providing some resistance to fungal infection. I evaluated traits such as spike length, spikelet number, spike density and spike inclination and their relationship with disease levels. Moderate heritability was estimated for spike traits, for example, with values of 0.66, 0.62 and 0.41 for spike length, spike density and spike inclination, respectively. Morphological and disease traits were generally negatively correlated, for instance, spike inclination was negatively correlated with DON, indicating that more inclined spike (parallel to soil surface) would have lower DON levels. This result agrees with the hypothesis that a more erect spike could keep water between florets for a longer period of time and consequently creating a microenvironment ideal for fungal development. The GWAS analysis identified

29 SNPs associated with morphological traits with values ranging from -0.0001 (spike density) to 9.58% (yield) of the trait mean. Three potential SNPs were identified for yield on chromosomes 1B, 3B and 6A, however multi-environment test is required to validate the results found in this study. Despite of the small effects observed for the spike traits, the use of traits such as spike length, spikelet number and spike inclination should be considered when phenotyping the population for FHB resistance.

In conclusion, this dissertation provided information about the phenotypic and genotypic response to increased temperature. I observed that important wheat QTL (*Fhb1, Rht, Vrn* and *Ppd*) did not respond well to increased temperature. Furthermore, small effect QTL can potentially reduce DON levels and traits such as spike length, spikelet number and spike inclination can potentially provide passive resistance to FHB.

BIBLIOGRAPHY

Agostinelli, A.M., A.J. Clark, G. Brown-Guedira, and D.A. Van Sanford. 2012. Optimizing phenotypic and genotypic selection for Fusarium head blight resistance in wheat. Euphytica 186:115–126. doi: 10.1007/s10681-011-0499-6

Allard, V., O. Veisz, B. Koszegi, M. Rousset, J.L. Gouis, and P. Martre. 2012. The quantitative response of wheat vernalization to environmental variables indicates that vernalization is not a response to cold temperature. J. Exp. Bot. 63:847–857. doi:10.1093/jxb/err316

Allen, M., O.P. Dube, W. Solecki, F. Aragon-Durand, W. Cramer, S. Humphreys, M. Kainuma, J. Kala, N. Mahowald, Y. Mulugetta, R. Perez, M. Wairiu and K. Zickfeld. 2018. Framing and Context. In: Global warming of 1.5°C. An IPCC Special Report on the impacts of global warming of 1.5°C above pre-industrial levels and related global greenhouse gas emission pathways, in the context of strengthening the global response to the treat of climate change, sustainable development, and efforts to eradicate poverty [V. Masson-Delmotte, P. Zhai, H.O. Portner, D. Roberts, J. Skea, P.R. Shukla, A. Pirani, W. Moufouma-Okia, C. Pean, R. Pidcock, S. Connors, J.B.R. Matthews, Y. Chen, X. Zhou, M.I. Gomis, E. Lonnoy, T. Maycock, M. Tignor, T. Waterfield (eds)]. In Press.

Arruda, M.P., P. Brown, G. Brown-Guedira, A.M. Krill, C. Thurber, K.R. Merrill, B.J. Foresman, and F.L. Kolb. 2016. Genome-wide association mapping of Fusarium head blight resistance in wheat using genotype-by-sequencing. The Plant Genome 9:1–14. doi: 10.3835/plantgenome2015.04.0028

Atlin, G.N., J.E. Cairns, and B. Das. 2017. Rapid breeding and varietal replacement are critical to adaptation of cropping systems in the developing world to climate change. Glob. Food Secur. 12:31–37. doi:10.1016/j.gfs.2017.01.008

Audenaert, K., E. Callewaert, M. Höfte, S. Saeger, and G. Haesaert. 2010. Hydrogen peroxide induced by fungicide prothioconazole triggers deoxynivalenol (DON) production by *Fusarium graminearum*. BMC Microbiology 10:112. doi: 10.1186/1471-2180-10-112

Audenaert, K., A. Vanheule, M. Höfte, and G. Haesaert. 2013. Deoxynivalenol: A major player in the multifaceted response of *Fusarium* to its environment. Toxins 6:1-19. doi: 10.3390/toxins6010001

Backhouse, D. 2914. Global distribution of *Fusarium graminearum*, *F. asiaticum* and *F. boothii* from wheat in relation to climate. Eur. J. Plant Pathol 139:161–173. doi:10.1007/s10658-013-0374-5

Bai, G., and G. Shaner. 2004. Management and resistance in wheat and barley to Fusarium head blight. Annu. Rev. Phytopathol. 42:135–161. doi: 10.1146/annurev.phyto.42.040803.140340

Balut, A.L. 2012. Validation of *Fhb1* and *QFhs.nau-2DL* in several soft red winter wheat populations. Ph.D. diss., University of Kentucky.

Balut, A.L., A.J. Clark, G. Brown-Guedira, E. Souza, and D.A. Van Sanford. 2013. Validation of *Fhb1* and *QFhs. nau-2DL* in several soft read winter wheat population. Crop Sci. 53:934–945. doi:10.2135/cropsci2012.09.0550

Bazakos, C., M. Hanemian, C. Trontin, J.M. Jimenez-Gomez, and O. Loudet. 2017. New strategies and tools in quantitative genetics: How to go from the phenotype to the genotype. Annu. Rev. Plant Biol. 68:435–455. doi: 10.1146/annurev-arplant-042916-040820

Begum, H., J.E. Spindel, A. Lalusin, T. Borromeo, G. Gregorio, J. Hernandez, P. Virk, B. Collard, S. R. McCouch. 2015. Genome-wide association mapping for yield and other agronomic traits in an elite breeding population of tropical rice (*Oryza sativa*). PLoS ONE 10(3):e0119873. doi: 10.1371/journal.pone.0119873

Bogard, M., M. Jourdan, V. Allard, P. Martre, M.R. Perretant, C. Ravel, E. Heumez, S. Orford, J. Snape, D. Griffiths, O. Gaju, J. Foulkes, and J.L. Gouis. 2011. Anthesis date mainly explained correlations between post-anthesis leaf senescence, grain yield, and grain protein concentration in a winter wheat population segregating for flowering time QTLs. Journal of Experimental Botany 68:3621–3636. doi: 10.1093/jxb/err061

Bonin, C.M., and F.L. Kolb. 2009. Resistance to *Fusarium* head blight and kernel damage in a winter wheat recombinant inbred line population. Crop Science 49:1304–1312. doi: 10.2135/cropsci2008.08.0459

Boonekamp, P.M. 2012. Are plant diseases too much ignored in the climate change debate? Eur J Plant Pathol 133:291–294. doi: 10.1007/s10658-011-9934-8

Borlaug, N.E., and C.R. Dowswell. 2013. Feeding a world of ten billion people: A 21st century challenge. In Proceedings of the International Congress “In the Wake of the Double Helix: From the Green Revolution to the Gene Revolution”, Bologna, Italy, 27–31.

Brachi, B., G.P. Morris, and J.O. Borevitz. 2011. Genome-wide association studies in plants: the missing heritability is in the field. Genome Biology 12:232. doi: 10.1186/gb-2011-12-10-232

Bradbury, P.J., Z. Zhang, D.E. Kroon, T.M. Casstevens, Y. Ramdoss and E.S. Buckler. 2007. TASSEL: software for association mapping of complex traits in diverse samples. Bioinformatics 23:2633–2635. doi:10.1093/bioinformatics/btm308

Buerstmayr, H., T. Ban, and J.A. Anderson. 2009. QTL mapping and marker-assisted selection for *Fusarium* head blight resistance in wheat: a review. Plant Breeding 128:1–26. doi: 10.1111/j.1439-0523.2008.01550.x

Buerstmayr, M., and H. Buerstmayr. 2015. Comparative mapping of quantitative trait loci for Fuarium head blight resistance and anther retention in the winter wheat population Capo x Arina. Theor Appl Genet 128:1519–1530. doi: 10.1007/s00122-015-2527-8

Buerstmayr, M., and H. Buerstmayr. 2016. The semidwarfing alleles *Rht-D1b* and *Rht-B1b* show marked differences in their associations with anther-retention in wheat heads and with Fusarium head blight susceptibility. Phytopathology 106:1544–1552. doi: 10.1094/PHYTO-05-16-0200-R

Buerstmayr, H., M. Lemmens, L. Hartl, L. Doldi, B. Steiner, M. Stierschneider, and P. Ruckenbauer. 2002. Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. I. Resistance to fungal spread (Type II resistance). Theor Appl Genet 104:84–91. doi: 10.1007/s001220200009

Buerstmayr, M., M. Lemmens, B. Steiner, and H. Buerstmayr. 2011. Advanced backcross QTL mapping of resistance to Fusarium head blight and plant morphological traits in a *Triticum macha* x *T. aestivum* population. Theor Appl Genet 123:293–306. doi: 10.1007/s00122-011-1584-x

Buerstmayr, H., B. Steiner, L. Hartl, M. Griesser, N. Angerer, D. Lengauer, T. Miedaner, B. Schneider, and M. Lemmens. 2003. Molecular mapping of QTLs for Fusarium head blight resistance in spring wheat. II. Resistance to fungal penetration and spread. Theor Appl Genet 107:503–508. doi: 10.1007/s00122-003-1272-6

Buerstmayr, H., Steiner, B., and Lemmens, M. 2010. Current knowledge on the genetics of Fusarium head blight resistance in wheat – Implications for resistance breeding. In: Canty, S., A. Clark, A. Anderson-Scully, E. Ellis, D. Van Sanford (eds). Proceeding of the 2010 National Fusarium Head Blight Forum, December 7-9, Milwaukee, WI.

Cai, J., S. Wang, T. Li, G. Zhang, and G. Bai. 2016. Multiple minor QTLs are responsible for *Fusarium* head blight resistance in Chinese wheat landrace Haiyanzhong. PLoS ONE 11, e0163292. doi:10.1371/journal.pone.0163292

Chakraborty, S., and A.C. Newton. 2011. Climate change, plant diseases and food security: an overview. Plant Pathology 60:2–14. doi: 10.1111/j.1365-3059.2010.02411

Chen, G.F., R.G. Wu, D.M. Li, H.X. Yu, Z. Deng, and J.C. Tian. 2017. Genomewide association study for seeding emergence and tiller number using SNP markers in an elite winter wheat population. J. Genet 96:177–186. doi: 10.1007/s12041-016-0731-1

Clark, A.J., D. Sarti-Dvorjak, G. Brown-Guedira, Y. Dong, B.-K.Baik, and D.A. Van Sanford. 2016 Identifying rare FHD-resistant segregants in intransigent backcross and F_2 winter wheat populations. Front. Microbiol. 7:1–14. doi:10.3389/fmicb.2016.00277.

Cowger, C., R. Weisz, C. Arellano, and P. Murphy. 2016. Profitability of integrated management of Fusarium head blight in North Caroline winter wheat. Phytopathology 106:814–823. doi: 10.1094/PHYTO-10-15-0263-R

Desjardins, A.E. 2003. *Gibberella* from A (venaceae) to Z (eae). Annual Review of Phytopathology 41:177–198. doi: 10.1146/annurev.phyto.41.011703.115501

De Vries, A.P. 1971. Flowering biology of wheat, particularly in view of hybrid seed production – review. Euphytica 20:152–170.

Diaz, A., M. Zikhali, A.S. Turner, P. Isaac, and D.A. Laurie. 2012. Copy number variation affecting the *Photoperiod-B1* and *Vernalization-A1* genes in associated with altered flowering time in wheat (*Triticum aestivum*). PLoS ONE 7(3): e33234. doi: 10.1371/journal.pone.0033234

Distelfeld, A., C. Li, and J. Dubcovsky. 2009. Regulation of flowering in temperate cereals. Current opinion in plant biology 12(2):178–184. doi: 10.1016/J.pbi.2008.12.010

Draeger, R., N. Gosman, A. Steed, E. Chandler, M. Thomsett, Srinivasachary, J. Schondelmaier, H. Buerstmayr, M. Lemmens, M. Schmolke, A. Mesterhazy, and P. Nicholson. 2007. Identification of QTLs for resistance to Fusarium head blight, DON accumulation and associated traits in the winter wheat variety Arina. Theor Appl Genet 115:617–625. doi: 10.1007/s00122-007-0592-3

Driedonks, N., I. Rieu, and W.H. Vriezen. 2016. Breeding for plant heat tolerance at vegetative and reproductive stages. Plant Reprod. 29:67–79. doi: 10.1007/s00497-016-0275-9

Eckardt, N.A. 2010. Evolution of domesticated bread wheat. Plant Cell 22(4):993. doi: 10.1105/tcp.110.220410.

Ehret, G.B. 2010. Genome-wide association studies: contribution of genomics to understanding blood pressure and essential hypertension. Curr Hypertens Rep. 12:17–25. doi: 10.1007/s11906-009-0086-6

Eichler, E.E., J. Flint, G. Gibson, A. Kong, S.M. Leal, J.H. Moore, and J.H. Nadeau. 2010. Missing heritability and strategies for finding the underlying causes of complex disease. Nat Rev Genet 11:446–450. doi: 10.1038/nrg2809

Emrich, K., F. Wilde, T. Miedaner, and H.P. Pielpho. 2008. REML approach for adjusting the *Fusarium* head blight rating to a phenological data in inoculated selection experiments of wheat. Theor Appl Genet 117:65–73. doi: 10.1007/s00122-008-0753-z

Falcone, D.S. 1960. *Introduction to Quantitative Genetics*, 1st ed.; Ronald Press: New York, NY, USA.

Falconer, D.S., and T.F. Mackay. 1996. Introduction to quantitative genetics. Essex, UK: Longman Group Ltd. pp.464.

Fang, S.B., K.Y. Tan, and S.X. Ren. 2010. Winter wheat yields decline with spring higher night temperature by controlled experiments. Sci. Agric. Sin. 43:3251–3258 [in Chinese, English abstract].

Fang, S.B., K.Y. Tan, S.X. Ren, X.S. Zhang, and J.F. Zhao. 2012. Fields experiments in North China show no decrease in winter wheat yields with night temperature increased by 2.0 – 2.5°C. Science China Earth Sceinces, 55:1021–1027. doi:10.1007/s11430-012-4404-5

FAO – Food and Agriculture Organization of the United Nations. 2019. http://www.fao.org/faostat/en/#data/QC (accessed 16 Feb. 2019)

FAO, IFAD, UNICEF, WFP and WHO. 2018. The State of Food Security and Nutrition in the World 2018. Building climate resilience for food security and nutrition. Rome, FAO. http://www.fao.org/3/I9553EN/i9553en.pdf

Farroq, M., H. Bramley, J.A. Palta, and K.H.M. Siddique. 2011. Heat stress in wheat during reproductive and grain-filling phases. Plant Science 30:491–507. doi: 10.1080/07352689.2011.615687

Fehr, W.R.1987. Principles of Cultivar Development, Vol.1, Theory and Technique, McGraw Hill Inc., New York.

Fels-Klerx, H.J., J.E. Olesen, M.S. Madsen, and P.W. Goedhart. 2012. Climate change increases deoxynivalenol contamination of wheat in north-western Europe. Food Additives & Contaminants: Part A 29:1593-1604. doi: 10.1080/19440049.2012.691555

Fels-Klerx, H.J.V., C. Liu, and P. Battilani. 2016 Modelling climate change impacts on mycotoxin contamination. *World Mycotoxin J. 9*:717–726. doi:10.3920/WMJ2016.2066

Ferris, R., R.H. Ellis, T.R. Wheeler, and P. Hadley. 1998. Effect of high temperature stress at anthesis on grain yield and biomass of field-grown crops of wheat. Ann Bot. 82:631–639. doi: 10.1006/anbo.1998.0740

Flint-Garcia, S.A., J.M. Thornsberry, and E.S. Buckler. 2003. Structure of linkage disequilibrium in plants. Annu. Rev. Plant Biol. 54:357–374. doi: 10.1146/annurev.arplant.54.031902.134907

Food and Drug Administration's. 2010. web site: https://www.fda.gov/Food/GuidanceRegulation/ucm120184.htm accessed: 08/18

Fuentes, R.G., H.R. Mickelson, R.H. Busch, R. Dill-Macky, C.K. Evans, W.G. Thompson, J.V. Wiersma, W. Xie, Y. Dong, and J.A. Anderson. 2005. Resource allocation and cultivar stability in breeding for Fusarium head blight resistance in spring wheat. Crop Sci. 45:1965-1972. doi: 10.2135/cropsci2004.0589

García, G.A., R.A. Serrago, F.G. González, G.A. Slafer, M.P. Reynolds, and D.J. Miralles. 2014. Wheat grain number: Identification of favourable physiological traits in an elite double-haploid population. Field Crop Research 168:126–134. doi: 10.1016/j.fcr.2014.07.018

George, A.W., and C. Cavanagh. 2015. Genome wide association mapping in plants. Theor Appl Genet. 128:1163–1174. doi: 10.1007/s00122-015-2497-x

Gervais, L., F. Dedryver, J.Y. Morlais, V. Bodusseau, S. Negre, M. Bilous, C. Groos, and M. Trottet. 2003. Mapping of quantitative trait loci for field resistance to Fusarium head blight in an European winter wheat. Theor Appl Genet 106:961–970. doi: 10.1007/s00122-002-1160-5

Giancaspro, A., S.L. Giove, D. Zito, A. Blanco, and A. Gadaleta. 2016. Mapping QTLs for Fusarium head blight resistance in an interspecific wheat population. Frontiers in Plant Science 7:1381. doi: 10.3389/fpld.2016.01381

Gilsinger, J., L. Kong, X. Shen, and H. Ohm. 2005. DNA markers associated with low Fusarium head blight incidence and narrow flower opening in wheat. Theor Appl Genet 110:1218–1225. doi: 10.1007/s00122-005-1953-4

Gomez, D., L. Vanzetti, M. Helguera, L. Lombardo, J. Fraschina, and D.J. Miralles. 2014. Effect of *Vrn-1*, *Ppd-1* genes and earliness *per se* on heading time in Argentinean bread wheat cultivars. Field Crop Research 158:73–81. doi: 10.1016/j.fcr.2013.12.023

Gooding, M.J., R.H. Ellis, P.R. Shewry, and J.D. Schofield. 2003. Effects of restricted water availability and increased temperature on the grain filling, drying and quality of winter wheat. J. Cereal Sci. 37:295–309. doi: 10.1006/jcrs.2002.0501

Gooding, R.W., H.N. Lafever, K.G. Campbell, and L.D. Herald. 1997. Registration of 'Freedom' wheat. Crop Science 37:1007.

Graham, S., and R.A. Browne. 2009. Anther extrusion and Fusarium head blight resistance in European wheat. J. Phytopathol 157:580–582. doi: 10.1111/j.1439-0434.2008.01524.x

Griffey, C.A., W.L. Rohrer, T.H. Pridgen, W.S. Brooks, J. Chen, J.A. Wilson, D. Nabati, D.E. Brann, E.G. Rucker, H.D. Behl, M.E. Vaughn, W.L. Sisson, T.R. Randall, R.A. Corbin, J.C. Kenner, D.W. Dunaway, R.M. Pitman, H.E. Bockelman, C. Gaines, D.L.

Long, D.V. McVey, S.E. Cambron, and L. Whitcher. 2005a. Registration of 'McCormick' wheat. Crop Science 45:417–419.

Griffey, C.A., W.L. Rohrer, T.H. Pridgen, W.S. Brooks, J. Chen, J.A. Wilson, D. Nabati, D.E. Brann, E.G. Rucker, H.D. Behl, M.E. Vaughn, W.L. Sisson, T.R. Randall, R.A. Corbin, J.C. Kenner, D.W. Dunaway, R.M. Pitman, A.E. Smid, H.E. Bockelman, C. Gaines, D.L. Long, D.V. McVey, S.E. Cambron, and L. Whitcher. 2005b. Registration of 'Tribute' wheat. Crop Sience 45:419–420.

Griffey, C.A., T.M. Starling, A.M. Price, W.L. Sisson, M.K. Das, T.H. Pridgen, M.E. Vaughn, W.L. Rohrer, and D.E. Brann. 2001. Registration of 'Roane' wheat. Crop Science 41:1359–1360. doi: 10.2135/cropsci2001.4141359x

Griffey, C.A., W.E. Thomason, R.M. Pitman, B.R. Beahm, J.J. Paling, J. Chen, J.K. Fanelli, J.C. Kenner, D.W. Dunaway, W.S. Brooks, M.E. Vaughn, E.G. Hokanson, H.D. Belh, R.A. Corbin, M.D. Hall, S. Liu, J.T. Custis, C.M. Waldenmaier, D.E. Starner, S.A. Gulick, S.R. Ashburn, D.L. Whitt, H.E. Bockelman, E.J. Souza, G.L. Brown-Guedira, J.A. Kolmer, D.L. Long, Y. Yin, X. Chen, and S.E. Cambron. 2010. Registration of 'Jamestown' wheat. J Plant Reg 4:28–33.

Griffiths, S., J. Simmonds, M. Leverington, Y. Wang, L. Fish, L. Sayers, L. Alibert, S. Orford, L. Wingen, L. Herry, S. Faure, D. Laurie, L. Bilham, and J. Snape. 2009. Meta-QTL analysis of the genetic control of ear emergence in elite European winter wheat germoplasm. Theor Appl Genet 119:383–395. doi: 10.1007/s00122-009-1046-x

Grogan, S.M., G. Brown-Guedira, S.C. Haley, G.S. McMaster, S.D. Reid, J. Smith, and P.F. Byrne. 2016. Allelic variation in developmental genes and effects on winter wheat heading date in the U.S. great plains. PLoS ONE 11(4): e0152852. doi: 10.1371/journal.pone.0152852

Guedira, M., M. Xiong, Y.F. Hao, J. Johnson, S. Herrison, D. Marshall, and G. Brown-Guedira. 2016. Heading date QTL in winter wheat (*Triticum aestivum* L.) coincide with major developmental genes VERNALIZATION1 and PHOTOPERIOD1. PLoS ONE 11(5): e0154242. doi: 10.1371/journl.pone.0154242

Hall, M.D. and D.A. Van Sanford. 2003. Diallel analysis of *Fusarium* head blight resistance in soft red winter wheat. Crop Sci. 43:1663–1670. doi:10.2135/cropsci2003.1663

He, X., M. Lillemo, J. Shi, J. Wu, Å. Bjørnstad, T. Belova, S. Dreisigacker, E. Duveiller, and P. Singh. 2016a. QTL characterization of Fusarium head blight resistance in CIMMYT bread wheat line Soru#1. PLoS ONE 11(6): e0158052. doi: 10.1371/journal.pone.0158052

He, X., P.K. Singh, S. Dreisigacker, S. Singh, M. Lillemo, and E. Duveiller. 2016b Dwarfing genes *Rht-B1b* and *Rht-D1b* are associated with both type I FHB susceptibility

and low anther extrusion in two bread wheat populations. PLoS ONE 11(9):e0162499. doi: 10.1371/journal.pone.0162499

Herndl, M., J.W. White, L.A. Hunt, S. Graeff, and W. Claupein. 2008. Field-based evaluation of vernalization requirement, photoperiod response and earliness *per se* in bread wheat (*Triticum aestivum* L.). Field Crop Research 105:193–201. doi: 10.1016/j.fcr.2007.10.002

Hilton, A.J., P. Jenkinson, T.W. Hollins, and D.W. Parry. 1999. Relationship between cultivar height and severity of *Fusarium* ear blight in wheat. Plant Pathol. 48:202–208. doi: 10.1046/j.1365-3059.1999.00339.x

Hitz, K. 2015. Breeding for Nitrogen Use Efficiency in Soft Red Winter Wheat. Master's Thesis, University of Kentucky, Lexington, KY, USA

Högy, P., C. Poll, S. Marhan, E. Kandeler, and A. Fangmeier. 2013. Impacts of temperature increase and change in precipitation pattern on crop yield and yield quality in barley. Food Chemistry 136:1470–1477. doi: 10.1016/j.foodchem.2012.09.056

Holland, J.B., and W.E. Nvquist. 2003. Cervantes-Martnez. Estimating and interpreting heritability for plant breeding: An update. Plant Breed. Rev. 22:9–112.

Hou, R., Z. Ouyang, Y. Li, G.V. Wilson, and H. Li. 2012. Is the change of winter wheat yield under warming caused by shortened reproductive period? Ecology and Evolution 2:2999–3008. doi: 10.1002.ece3.403

Huang, X., and B. Han. 2014. Natural variations and genome-wide association studies in crop plants. Plant Biol. 65:531–551. doi: 10.1146/annurev-arplant-050213-035715

Intergovernmental Panel on Climate Change (IPCC). 2014. Synthesis Report. Contribution of Working Groups I, II, and II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change; Core Writing Team, Pachauri, R.K., L.A. Meyer, Eds.; IPCC: Geneva, Switzerland.

International Wheat Genome Sequencing Consortium (IWGSC). 2018. Shifting the limits in wheat research and breeding using a fully annotated reference genome. Science 361:6403. doi: 10.1126/science.aar7191

Islam, M.S., G. Brown-Guedira, D. Van Sanford, H. Ohm, Y. Dong, and A.L. McKendry. 2016. Novel QTL associated with Fusarium head blight resistance in Truman soft red winter wheat. Euphytica 207:571–592. doi: 10.1007/s10681-015-1550-9

Jannink, J.L. 2007. Identifying quantitative trait locus by genetic background interactions in association studies. Genetics 176:553–561. doi: 10.1534/genetics.106.062992

Jia, H., J. Zhou, S. Xue, G. Li, H. Yan, C. Ran, Y. Zhang, J. Shi, L. Jia, X. Wang, J. Luo, and Z. Ma. 2017. A journey to understand wheat Fusarium head blight resistance in the Chinese wheat landrace Wangshibai. The Crop Journal 6:48–59. doi: 10.1016/j.cj.2017.09.006

Jiang G.L., Y. Dong, J.R. Shi, and R.W. Ward. 2007a. QTL analysis of resistance to Fusarium head blight in the novel wheat germplasm CJ 9306. II. Resistance to deoxynivalenol accumulation and grain yield loss. Theor Appl Genet 115:1043–1052. doi: 10.1007/s00122-007-0630-1

Jiang G.L., J.R. Shi, and R.W. Ward. 2007b. QTL analysis of resistance to Fusarium head blight in the novel wheat germplasm CJ 9306. I. Resistance to fungal spread. Theor Appl Genet 116:3–13. doi: 10.1007/s.00122-007-0641-y

Jiang, Y., Y. Zhao, B. Rodemann, J. Plieske, S. Kollers, V. Korzum, E. Ebmeyer, O. Argillier, M. Hinze, J. Ling, M.S. Roder, M.W. Ganal, M.F. Mette, and J.C. Reif. 2015. Potential and limits to unravel the genetic architecture and predict the variation of Fusarium head blight resistance in European winter wheat (*Triticum aestivum* L.). Heredity 114:318–326. doi: 10.1038/hdy.2014.104

Jin, F., D. Zhang, W. Bockus, P.S. Baenziger, B. Carver, and G. Bai. 2013. Fusarium head blight resistance in U.S. winter wheat cultivars and elite breeding lines. Crop Science 53:2006–2013. doi: 10.2135/cropsci2012.09.0531

Johnson, D.D., G.K. Flaskerud, R.D. Taylor, and V. Satyanarayana. 2003. Quantifying economic impacts of Fusarium head blight in wheat. In: Leonard, K.J., and W.R. Bushnell. (eds) Fusarium head blight of wheat and barley. APS Press, St. Paul, MN, pp 461-483.

Jones, S., A. Farooqi, J. Foulkes, D.L. Sparkes, R. Linforth, and R.V. Ray. 2018. Canopy and ear traits associated with avoidance of Fusarium head blight in wheat. Frontiers in Plant Science 9:1021. doi: 10.3389/fpls.2018.01021

Kazan, K., and D.M. Gardiner. 2017. Transcriptomics of cereal-*Fusarium graminearum* interactions: what we have learned so far. Molecular Plant Pathology. doi:10.1111/mmp.12561

Kiseleva, A.A., A.B. Shcherban, I.N. Leonova, Z. Frenkel, and E.A. Salina. 2016. Identification of new heading date determinants in wheat 5B chromosome. BMC Plant Biol. 35-81. doi: 10.1186/s12870-015-0688-x

Klahr, A., G. Zimmermann, G. Wenzel, and V. Mohler. 2007. Effects of environment, disease progress, plant height and heading date on the detection of QTLs for resistance to Fusarium head blight in an European winter wheat cross. Euphytica 154:17–28. doi: 10.1007/s10681-006-9264-7

Knapp, S.J., W.W. Stroup, and W.M. Ross. 1985. Exact confidence intervals for heritability on a progeny mean basis. *Crop Sci.* *25*:92–195. doi:10.2135/cropsci1985.0011183X002500010046x

Kollers, S., B. Rodemann, J. Ling, V. Korzun, E. Ebmeyer, O. Argillier, M. Hinze, J. Plieske, D. Kulosa, M.W. Ganal, and M.S. Röder. 2013. Whole genome association mapping of *Fusarium* head blight resistance in European winter wheat (*Triticum aestivum* L.). PLoS ONE 8(2): e575000. doi: 10.1371/journal.pone.0057500

Korte, A., and A. Farlow. 2013. The advantages and limitations of trait analysis with GWAS: a review. Plant Methods 9:29. doi: 10.1186/1746-4811-9-29

Kristensen, P.S., A. Jahoor, J.R. Andersen, F. Cericola, J. Orabi, L.J. Janss, and J. Jensen. 2018. Genome-wide association studies and comparison of models and cross-validation strategies for genomic prediction of quality traits in advanced winter wheat breeding lines. Frontiers in Plant Science 9:69. doi: 10.3389/fpls.2018.00069

Kubo, K., M. Fujita, N. Kawada, T. Nakajima, K. Nakamura, H. Maejima, T. Ushiyama, K. Hatta, and H. Matsunaka. 2013. Minor differences in anther extrusion affect resistance to Fusarium head blight in wheat. J. Phytopathol 161:308–314. doi: 10.1111/jph.12060

Kugler, K.G., G. Siegwart, T. Nussbaumer, C. Ametz, M. Spannagl, B. Steiner, M. Lemmens, K.F.X. Mayer, H. Buerstmayr, and W. Schweiger. 2013. Quantitative trait loci-dependent analysis of a gene co-expression network associated with Fusarium head blight resistance in bread wheat (*Triticum aestivum* L.). BMC Genomics 14:728. doi: 10.1186/1471-2164-14-728

Law, C.N., A.J. Worland, and B. Giorgi. 1976. The genetic control of ear-emergence time by chromosomes 5A and 5D of wheat. Heredity 36:49–58. doi: 10.1038/hdy.1976.5

Lee, S.J., S.H. Ban, G.H. Kim, S.I. Kwon, J.H. Kim, and C. Choi. 2017. Identification of potential gene-associated major traits using GBS-GWAS for Korean apple germplasm collections. Plant Breed. 1–10. doi:10.1111/pbr.12544

Lesk, C., P. Rowhani, and N. Ramankutty. 2016. Influence of extreme weather disasters on global crop production. Nature 529:84–87. doi: 10.1038/nature16467

Li, X., D. Jiang, and F. Liu. 2016. Soil warming enhances the hidden shift of elemental stoichiometry by elevated CO_2 in wheat. Science Report 6:23313. doi: 10.1038/srep23313

Lipka, A.E., F. Tian, Q. Wang, J. Peiffer, M. Li, P.J. Bradbury, M.A. Gore, E.S. Buckler, and Z. Zhang. 2012. GAPIT: Genome association and prediction integrated tool. Bioinformatics 28:2397–2399. doi:10.1093/bioinformatics/bts444

Liu, B., S. Asseng, C. Müller, F. Ewert, J. Elliott, D.B. Lobell, P. Martre, A.C. Ruane, D. Wallach, J.W. Jones, et al. 2016. Similar estimates of temperature impacts on global wheat

yield by three independent methods. Nature Climate Change 6:1130–1136. doi:10.1038/nclimate3115

Liu, S., Z.A. Abate, H. Lu, T. Musket, G.L. Davis, and A.L. McKendry. 2007. QTL associated with Fusarium head blight resistance in the soft red winter wheat Ernie. Theor Appl Genet 115:417–427. doi: 10.1007/s00122-007-0577-2

Liu, S., M.D. Christopher, C.A. Griffey, M.D. Hall, P.G. Gundrum, and W.S. Brooks. 2012. Molecular characterization of resistance to Fusarium head blight in U.S. soft red winter wheat breeding line VA00W-38. Crop Science 52:2283–2292. doi: 10.2135/cropsci2012.03.0144

Liu, S., C.A. Griffey, M.D. Hall, A.L. McKendry, J. Chen, W.S. Brooks, G. Brown-Guedira, D.A. Van Sanford, and D.G. Schmale. 2013. Molecular characterization of field resistance to Fusarium head blight in two US soft red winter wheat cultivars. Theor Appl Genet. 126:2485–2498. doi: 10.1007/s00122-013-2149-y

Liu, S., M.D. Hall, C.A. Griffey, and A.L. McKendry. 2009. Meta-analysis of QTL associated with Fusarium head blight resistance in wheat. Crop Science 49:1995–1968. doi: 10.2135/cropsci2009.03.0115

Liu, K., X. Sun, T. Ning, X. Duan, Q. Wang, T. Liu, Y. An, X. Guan, J. Tian, and J. Chen. 2018. Genetic dissection of wheat panicle traits using linkage analysis and a genome-wide association study. Theor Appl Genet 131:1073–1090. doi: 10.1007/s00122-018-3059-9

Lobell, D.B., J.I. Ortiz-Monasterio, G.P. Asner, P.A. Matson, R.L. Naylor, and W.P. Falcon. 2005. Analysis of wheat yield and climatic trends in Mexico. Field Crops Res. 94:250–256. doi: 10.1016/j.fcr.2005.01.007

Lu, Q., M. Lillemo, H. Skinnes, X. He, J. Shi, F. Ji, Y. Dong, and Å. Bjørnstad. 2013. Anther extrusion and plant height are associated with Type I resistance to Fusarium head blight in bread wheat line 'Shanghai-3/Catbird'. Theor Appl Genet. 126:317–334. doi: 10.1007/s00122-012-1981-9

Lu, Q., A. Szabo-Hever, Å. Bjørnstad, M. Lillemo, K. Semagn, A. Mesterhazy, F. Ji, J. Shi, and H. Skinnes. 2011. Two major resistance quantitative trait loci are required to counteract the increased susceptibility to Fusarium head blight of the *Rht-D1b* dwarfing gene in wheat. Crop Science 51:2430–2438. doi: 10.2135/cropsci2010.12.0671

Machado, A.K., N.A. Brown, M. Urban, K. Kanyuka, and K.E. Hammond-Kosack. 2017. RNAi as an emerging approach to control Fusarium head blight disease and mycotoxin contamination in cereals. Pest Manag Sci 74:790–799. doi: 10.1002/ps.4748

Mamo, B.E., and B.J. Steffenson. 2015. Genome-wide association mapping of Fusarium head blight resistance and agromorphological traits in barley landraces from Ethiopia and Eritrea. Crop Sci. 55:1494–1512. doi: 10.2135/cropsci2014.06.0428

Mao, L.-S., Y.-M. Wei, W. Cao, X.-J. Lan, M. Yu, Z.-M. Chen, G.-Y. Chen, and Y.-L. Zheng. 2010. Confirmation of the relationship between plant height and Fusarium head blight resistance in wheat (*Triticum aestivum* L.) by QTL meta-analysis. Euphytica 174:343–356. doi: 10.1007/s10681-010-0128-9

Massman, J., B. Cooper, R. Horsley, S. Neate, R. Dill-Macky, S. Chao, Y. Dong, P. Schwarz, G.J. Muehlbauer, and K.P. Smith. 2011. Genome-wide association mapping of Fusarium head blight resistance in contemporary barley breeding germplasm. Mol Breeding 27:439–454. doi: 10.1007/s11032-010-9442-0

McKendry, A.L., J.E. Berg, D.N. Tague, and K.D. Kephart. 1995. Registration of Ernie wheat. Crop Science 35:1513

McKendry, A.L., D.N. Tague, R.L. Wright, J.A. Tremain, and S.P. Conley. 2005. Registration of 'Truman' wheat. Crop Science 45:421–423

McKendry, A.L., D.N. Tague, R.L. Wright, and J.A. Tremain. 2007. Registration of 'Bess' wheat. J Plant Reg 1:21–23

McMullen, M., G. Bergstrom, E. Wolf, R. Dill-Macky, D. Hershman, G. Shaner, and D. Van Sanford. 2012. A unified effort to fight an enemy of wheat and barley: Fusarium head blight. Plant Disease 96:1712–1728. doi: 10.1094/PDIS-03-12-0291-FE

Meister, G., and T. Tuschl. 2004. Mechanisms of gene silencing by double-stranded RNA. Nature 431:343–349. doi: 10.1038/nature02873

Mesterházy, A., T. Bartók, C.G. Mirocha, and R. Komoróczy. 1999. Nature of wheat resistance to *Fusarium* head blight and the role of deoxynivalenol for breeding. Plant Breeding 118:97–110. doi: 10.1046/j.1439-0523.1999.118002097.x

Mesterhazy, M., R. Jones, and D. Gallenberg. 1997. Scab of wheat and barley: A re-emerging disease of devasting impact. Plant Dis. 81:1340–1348. doi:10.1094/PDIS.1997.81.12.1340

Miedaner, T., and V. Korzun. 2012. Marker-assisted selection for disease resistance in wheat and barley breeding. Phytopathology 6:561–566. doi: 10.1094/PHYTO-05-11-0157

Miedaner, T., T. Wurschum, H.P. Maurer, V. Kirzum, E. Ebmeyer, and J.C. Reif. 2011. Association mapping for Fusarium head blight resistance in European soft winter wheat. Mol Breeding 28:647–655. doi: 10.1007/s11032-010-9516-z

Mirocha, C.J., E. Kolaczkowski, W. Xie, H. Yu, and H. Jelen. 1998. Analysis of deoxynivalenol and its derivatives (batch and single) using gas chromatography/mass spectrometry. J. Agric. Food Chem. 46:1414–1418 doi:10.1021/jf9708570

Mitchell, R.A.C., V.J. Mitchell, S.P. Driscoll, J. Franklin, and D.W. Lawlor. 1993. Effects of increased CO_2 concentration and temperature on growth and yield of winter wheat at two levels of nitrogen application. Plant, Cell & Environment 16:521–529. doi: 10.1111/j.1365-3040.1993.tb00899.x

Muqaddasi, Q.H., J. Brassac, A. Börner, K. Pillen, and M.S. Röder. 2017. Genetic architecture of anther extrusion in spring and winter wheat. Frontiers in Plant Science 8:754. doi: 10.3389/fpls.2017.00754

Myles, S., J. Peiffer, P.J. Brown, E.S. Ersoz, Z. Zhang, D.E. Costich, and E.S. Buckler. 2009. Association mapping: critical considerations shift from genotyping to experimental desing. The Plant Cell 21:2194–2202. doi: 10.1105/tcp.109.068437

Nganje, W.E., D.A. Bangsund, F.L. Leistritz, W.W. Wilson, and N.M. Tiapo. 2002. Estimating the economic impact of a crop disease: the case of fusarium head blight in U.S. wheat and barley. In: Canty, S., J. Lewis, L. Silver, and R. Ward. (eds) Proceedings of the 2002 National Fusarium head blight forum, December 7-9, Erlanger, KY

Ogbonnaya, F.C., A. Rasheed, E.C. Okechukwu, A. Jighly, F. Makdis, T. Wuletaw, A. Hagras, M.I. Uguru, and C.U. Agbo. 2017. Genome-wide association study for agronomic and physiological traits in spring wheat evaluated in a range of heat prone environments. Theor Appl Genet 130:1819–1835. doi: 10.1007/s00122-017-2927-z

Ohm, H.W., G. Shaner, R. Ratcliffe, D. Huber, H. Sharma, K. Perry, G. Buechley, and S. Cambron. 2000. Registration of 'Godlfield' wheat. Crop Science 40:581–582. doi: 10.2135/cropsci2000.0031rcv

Paillard, S., T. Schnurbusch, R. Tiwari, M. Messmer, M. Winzeler, B. Keller, and G. Schachermayr. 2004. QTL analysis of resistance to Fusarium head blight in Swiss winter wheat (*Triticum aestivum* L.). Theor Appl Genet 109:323–332. doi: 10.1007/s00122-004-1628-6

Parry, D.W., P. Jenkinson, and L. McLeod. 1995. *Fusarium* ear blight (scab) in small grain cereals – a review. Plant Pathology 44:207–238. doi: 10.1111/j.1365-3059-1995.tb02773.x

Pask, A., A.K. Joshi, Y. Manès, I. Sharma, R. Chatrath, G.P. Singh, V.S. Sohu, G.S. Mavi, V.S.P. Sakuru, I.K. Kalappanavar, V.K. Mishra, B. Arun, M.Y. Mujahid, M. Hussain, N.R. Gautam, N.C.D. Barma, A. Hakim, W. Hoppitt, R. Trethowan, and M.P. Reynolds. 2014. A wheat phenotyping network to incorporate physiological traits for climate change in South Asia. Field Crops Res *168*:156–167. doi:10.1016/j.fcr.2014.07.004

Patil, R.H., M. Laegdsmand, J.E. Olesen, and J.R. Porter. 2010Growth and yield response of winter wheat to soil warming and rainfall patterns. J. Agric. Sci. *148*:553–566. doi:10.1017/S0021859610000419

Petersen, S., J.H. Lyerly, P.V. Maloney, G. Brown-Guedira, C. Cowger, J.M. Costa, Y. Dong, and J.P. Murphy. 2016. Mapping of *Fusarium* head blight resistance quantitative trait loci in winter wheat cultivar NC-Neuse. Crop Science 56:1473–1483. doi: 10.2135/cropsci2015.05.0312

Prasad, P.V.V., S.R. Pisipati, Z. Ristic, U. Bukovnik, and A.K. Fritz. 2008. Impact of nighttime temperature on physiology and growth of spring wheat. Crop Sci. 48:2372–2380. doi: 10.2135/cropsci2007.12.0717

Qu, A.-L., Y.-F. Ding, Q. Jiang, and C. Zhu. 2013. Molecular mechanisms of the plant heat stress response. Biochemical and Biophysical Research Communications 432:203–207. doi: 10.1016/j.bbrc.2013.01.104

Quintero, A.; G. Molero, M.P. Reynolds, and D.F. Calderini. 2018. Trade-off between grain weight and grain number in wheat depends on GxE interaction: A case study of an elite CIMMYT panel (CIMCOG). European Journal of Agronomy 92;17-29. doi: 10.1016/j.eja.2017.09.007

R Core Team. 2013. R: A language and environment for statistical computing. R foundation for statistical computing, Vienna, Austria. www.R-project.org/.

Rawat, N., M.O. Pumphrey, S. Liu, X. Zhang, V.K. Tiwari, K. Ando, H.N. Trick, W.W. Bockus, E. Akhunov, J.A. Anderson, and B.S. Gill. 2016. Wheat *Fhb1* encodes a chimeric lectin with agglutinin domains and a pore-forming toxin-like domain conferring resistance to Fusarium head blight. Nature Genetics 48:1576–1580. doi: 10.1038/ng.3706

Ray, D.K., N.D. Mueller, P.C. West, and J.A. Foley. 2013 Yield trends are insufficient to double global crop production by 2050. PLoS ONE 8, e66428. doi:10.1371/journal.pone.0066428

Reis, E.M., and M.A. Carmona. 2013. Integrated disease management of Fusarium head blight, in *Fusarium* head blight in Latin America, ed. by T.M. Alconada Magliano, S.N. Chulze. Springer, Dordrecht, pp. 159–173

Rezaei, E.E., S. Siebert, and F. Ewert. 2015. Intensity of heat stress in winter wheat – phenology compensates for adverse effect of global warming. Environ. Res. Lett. 10:024012. doi: 10.1088/1748-9326/10/2/024012

Robertson, A. 1967. The nature of quantitative genetic variation. In *Heritage from Mendel*; Brink, A., Ed.; University of Wisconsin: Madison, WI, USA, pp. 265–280

Rosenzweig, C., J. Elliott, D. Deryng, A.C. Ruane, C. Müller, A. Arneth, K.J. Boote, C. Folberth, M. Glotter, N. Khabarov, K. Neumann, F. Piontek, T.A.M. Pugh, E. Schmid, E. Stehfest, H. Yang, and J.W. Jones. 2014. Assessing agricultural risk of climate change in the 21st century in a global gridded crop model intercomparison. PNAS 111:3268–3273. doi: 10.1073/pnas.1222463110

Ruelland, E., and A. Zachowski. 2010. How plants sense temperature. Environ. Exper. Bot. 69:225–232. doi: 10.1016/j.envexpbot.2010.05.011

Russell, K. 2017. Genotype × Environment × Management: Implications for Selection to Heat Stress Tolerance and Nitrogen Use Efficiency in Soft Red Winter Wheat. Ph.D. Thesis, University of Kentucky, Lexington, KY, USA

Salgado, J.D., L.V. Madden, and P.A. Paul. 2015. Quantifying the effects of Fusarium head blight on grain yield and test weight in soft red winter wheat. Phytopathology 105:295–306 . doi: 10.1094/PHYTO-08-14-0215-R

SAS Institute. *The SAS System for Windows™, Release 9.3*; SAS Institute Inc.: Cary, NC, USA. 2011

SAS Institute. JMP for Macintosh™, Release 13.2. SAS Institute Inc. Cary, NC, USA, 2018.

Saville, R.J., N. Gosman, C.J. Burt, J. Makepeace, A. Steed, M. Corbitt, E. Chandler, J.K.M. Brown, M.I. Boulton, and P. Nicholson. 2012. The 'green revolution' dwarfing genes play a role in disease resistance in *Triticum aestivum* and *Hordeum vulgare*. Journal of Experimental Botany 63:1271–1283. doi: 10.1093/jxb/err350

Schroeder, H.W., and J.J. Christensen. 1963. Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. Phytopathology 53:831–838.

Schmid, M., and J. Bennewitz. 2017. Invited review: Genome-wide association analysis for quantitative traits in livestock – a selective review of statistical models and experimental designs. Arch. Anim. Breed. 60:335-346, doi: 10.5194/aab-60-335-2017

Schulthess, A.W., Y. Zhao, C.F.H. Longin, and J.C. Reif. 2018. Advantages and limitations of multiple-trait genomic prediction for *Fusarium* head blight severity in hybrid wheat (*Triticum aestivum* L.). Theor App Genet 131:685–701. doi:10.1007/s00122-017-3029-7

Semagn, K., H. Skinner, Å. Bjørnstad, A.G. Marøy, and Y. Tarkegne. 2007. Quantitative trait loci controlling Fusarium head blight resistance and low deoxynivalenol content in hexaploid wheat population from 'Arina' and NK93604. Crop Science 47:294–303. doi: 10.2135/cropsci2006.02.0095

Shen, X., M. Ittu, and H.W. Ohm. 2003. Quantitative trait loci conditioning resistance to Fusarium head blight in wheat line F201R. Crop Science 43:850–857. doi: 10.2135/cropsci2003.8500

Sinclair, T.R., and P.D. Jamieson. 2006. Grain number, wheat yield, and bottling beer: An analysis. Field Crop Research 98:60-67. doi: 10.1016/j.fcr.2005.12.006

Skelsey, P., and A.C. Newton. 2015. Future environmental and geographic risks of *Fusarium* head blight of wheat in Scotland. Eur J Plant Pathol 142:133–147. doi: 10.1007/s10658-015-0598-7

Skinnes, H., K. Semagn, Y. Tarkegne, A.G. Marøy, and Å. Bjørnstad. 2010. The inheritance of anther extrusion in hexaploidy wheat and its relationship to *Fusarium* head blight resistance and deoxynivalenol content. Plant Breeding 129:149–155. doi: 10.1111/j.1439-0523.2009.01731.x

Snape, J.W., K. Butterworth, E. Whitechurch, A.J. Worland. 2001. Waiting for fine times: genetics of flowering time in wheat. Euphytica 119: 185-190. doi: 10.1023/A:1017594422176

Spanic, V., M.V. Vuletic, G.Drezner, Z. Zdunic, and D. Horvat. 2017. Performance indices in wheat chlorophyll *a* fluorescence and protein quality influenced by FHB. Pathogens 6:59. doi: 10.3390/pathogens6040059

Srinivasachary, N. Gosman, A. Steed, T.W. Hollins, R. Bayles, P. Jennings, and P. Nicholson. 2009. Semi-dwarfing *Rht-B1* and *Rht-D1* loci of wheat differ significantly in their influence on resistance to Fusarium head blight. Theor Appl Genet. 118:695–702. doi: 10.1007/s00122-008-0930-0

Srinivasachary, N. Gosman, A. Steed, J. Simmonds, M. Leverington-Waite, Y. Wang, J. Snape, and P. Nicholson. 2008. Susceptibility to *Fusarium* head blight is associated with the *Rht-D1b* semi-dwarfing allele in wheat. Theor Appl Genet. 116:1145–1153. doi: 10.1007/s00122-008-0742-2

Steiner, B., M. Buerstmayr, S. Michel, W. Schweiger, M. Lemmens, and H. Buerstmayr. 2017. Breeding strategies and advances in line selection for Fusarium head blight resistance in wheat. Trop. Plant Pathol. 42:165–174. doi: 10.1007/s40858-017-0127-7

Steiner, B., M. Lemmens, M. Griesser, U. Scholz, J. Schondelmaier, and H. Buerstmayr. 2004. Molecular mapping of resistance to Fusarium head blight in the spring wheat cultivar Frontana. Theor Appl Genet 109:215–224. doi: 10.1007/s00122-004-1620-1

Suzuki, T., M. Sato, and T. Takeuchi. 2012. Evaluation of the effects of five QTL regions on *Fusarium* head blight resistance and agronomic traits in spring wheat (*Triticum aestivum* L.). Breeding Science 62:11–17. doi: 10.1270/jsbbs.62.11

Tack, J., A. Barkley, and L.L. Nalley. 2015. Effect of warming temperatures on US wheat yields. PNAS 112:6931–6936. doi: 10.1073/pnas.1415181112

Tataw, J.T., F. Baier, F. Krottenthaler, B. Pachler, E. Schwaiger, S. Wyhlidal, H. Formayer, J. Hösch, A. Baumgarten, and J.G. Zaller. 2016. Climate change induced rainfall patterns affect wheat productivity and agroecosystem functioning dependent on soil types. Ecol Res 31:203–212. doi: 10.1007/s11284-015-1328-5

Tessmann, E.W., and D.A. Van Sanford. 2018. GWAS for Fusarium head blight related traits in winter wheat (*Triticum aestivum* L.) in an artificially warmed treatment. Agronomy 8:68. doi: 10.3390/agronomy8050068

Tian, Y., C. Zheng, J. Chen, C. Chen, A. Deng, Z. Song, B. Zhang, and W. Zhang. 2014. Climatic warming increases winter wheat yield but reduces grain nitrogen concentration in East China. PLoS ONE 9(4): e95108. doi:10.1371/journal.pone.0095108

Trevaskis, B. 2010. The central role of the *VERNALIZATION1* gene in the vernalization response of cereals. Functional Plant Biology 37(6):479–487. doi: 10.1071/FP10056

Turuspekov, Y., A. Baibulatova, K. Yermekbayev, L. Tokhetova, V. Chudinov, G. Sereda, M. Ganal, S. Griffiths, S. Abugalieva. 2017. GWAS for plant growth stages and yield components in spring wheat (*Triticum aestivum*) harvested in three regions of Kazakhstan. BCM Plant Biology 17:190. doi: 10.1186/s12870-017-1131-2

United Nations. World population prospects: The 2017 Revision. https://www.un.org/development/desa/publications/word-population-prospects-the-2017-revision.html (accessed 31 May 2018).

Urrea, C.A., R.D. Horsley, B.J. Steffenson, and P.B. Schwarz. 2002. Heritability of Fusarium head blight resistance and deoxynivalenol accumulation from barley accession CIho 4196. Crop Science 42:1404–1408. doi: 10.2135/cropsci2002.1404

Vaughan, M., D. Backhouse, and E.M. Del Ponte. 2016. Climate change impacts on the ecology of *Fusarium graminearum* species complex and susceptibility of wheat to *Fusarium* head blight: a review. World Mycotoxin Journal 9 (5):685-700. doi: 10.3920/WMJ2016.2053

Visscher, P.M., M.A. Brown, M.I. McCarthy, and J. Yang. 2012. Five years of GWAS discovery. The American Journal of Human Genetics 90:7–24. doi: 10.1016/j.ajhg.2011.11029

Wang, S.-X., Y.-L. Zhu, D.-X. Zhang, H. Shao, P. Liu, J.-B. Hu, H. Zhang, H.-P. Zhang, C. Chang, J. Lu, X.-C. Xia, G.-L. Sun, and C.-X. Ma. 2017. Genome-wide association study for grain yield and related traits in elite wheat varieties and advanced line using SNP markers. PLoS ONE 12(11): e0188662. doi: 10.1371/journal.pone.0188662

Worland, A.J., A. Börner, V. Korzun, W.M. Li, S. Petrovic, and E.J. Sayers. 1998. The influence of photoperiod genes on the adaptability of European winter wheats. Euphytica 100:385–394. doi: 10.1023/A:1018327700985

Worland, T., and J.W. Snape. 2001. Genetic basis of worldwide wheat varietal improvement. In: Bonjean, A.P., W.J. Angus, eds. The world wheat book: a history of wheat breeding, Paris: Lavoisier Publishing, pp. 59-100

Wu, X., H. Liu, X. Li, Y. Tian, and M.D. Mahecha. 2017 Response of winter wheat yields to warming-mediated vernalization variations across temperature Europe. Front. Ecol. Evol. *5*:1–10, doi:10.3389/fev0.2017.00126

Xiao, Y., H. Liu, L. Wu, M. Warburton, and J. Yan. 2017. Genome-wide association studies in maize: praise and stargaze. Molecular Plant 10:359–374. doi: 10.1016/j.molp.2016.12.008

Xue, S., G. Li, H. Jia, F. Xu, F. Lin, M. Tang, Y. Wang, X. An, H. Xu, L. Zhang, Z. Kong, and Z. Ma. 2010. Fine mapping *Fhb4*, a major QTL conditioning resistance to Fusarium infection in bread wheat (*Triticum aestivum* L.). Theor Appl Genet 121:147–156. doi: 10.1007/s00122-010-1298-5

Xue, S., F. Xu, M. Tang, Y. Zhou, G. Li, X. An, F. Lin, H. Xu, H. Jia, L. Zhang, Z. Kong, and Z. Ma. 2011. Precise mapping *Fhb5*, a major QTL conditioning resistance to Fusarium infection in bread wheat (*Triticum aestivum* L.). Theor Appl Genet 123:1055–1063. doi: 10.1007/s00122-011-1647-z

Yan, W., H.B. Li, S.B. Cai, H.X. Ma, G.J. Rebetzke, and C.J. Liu. 2011. Effects of plant height on type I and type II resistance to fusarium head blight in wheat. Plant Pathology 60:506–512. doi: 10.1111/j.1365-3059-2011-02426.x

Yi, X., J. Cheng, Z. Jiang, W. Hu, T. Bie, D. Gao, D. Li, R. Wu, Y. Li, S. Chen, X. Cheng, J. Liu, Y. Zang, and S. Cheng. 2018. Genetic analysis of Fusarium head blight resistance in CIMMYT bread wheat line C615 using traditional and conditional QTL mapping. Frontiers in Plant Science 9:573. doi: 10.3389/fpls.2018.00573

Yu, J., G. Pressoir, W.H. Briggs, I.V. Bi, M. Yamasaki, J.F. Doebley, M.D. McMullen, B.S. Gaut, D.M. Nielsen, J.B. Holland, S. Kresovich and E.S. Buckler. 2006. A unified mixed-model method for association mapping that accounts for multiple levels of relatedness. Nature genetics 38:203–208. doi: 10.1038/ng1702

Zanke, C., J. Ling, J. Plieske, S. Kollers, E. Ebmeyer, V. Korzun, O. Aguillier, G. Stiewe, M. Hinze, S. Beier, M. W. Ganal, and M.S. Röder. 2014. Genetic architecture of main effect QTL for heading date in European winter wheat. Frontiers in Plant Science 5:217. doi: 10.2289/fpls.2014.00217

Zhang, X.-W., L.-J. Jia, Y. Zhang, G. Jiang, X. Li, D. Zhang, and W.-H. Tang. 2012. In planta stage-specific fungal gene profiling elucidates the molecular strategies of *Fusarium graminearum* growing inside wheat coleoptiles. The Plant Cell. doi: 01.1105/tpc.112.105957

Zhao, C.; B. Liu, S. Piao, X. Wang, D.B. Lobell, Y. Huang, M. Huang, Y. Yao, S. Bassu, P. Ciais, J.-L. Durand, J. Elliott, F. Ewert, I.A. Janssens, T. Li, E. Lin, Q. Liu, P. Martre, C. Müller, S. Peng, J. Peñuelas, A.C. Ruane, D. Wallach, T. Wang, D. Wu, Z. Liu, Y. Zhu,

Z. Zhu, and S. Asseng. 2017. Temperature increase reduces global yields of major crops in four independent estimates. PNAS 114:9326–9331. doi: 10.1073/pnas.1701762114

Zheng, B., K. Chenu, M.F. Dreccer, and S.C. Chapman. 2012. Breeding for the future: what are the potential impacts of future frost and heat events on sowing and flowering time requirements for Australian bread wheat (*Triticum aestivum*) varieties? Global Change Biology 18:1899–2914.doi: 10.1111/j.1365-2486.2012.02724.x

Zheng, B., K. Chenu, and S.C. Chapman. 2016. Velocity of temperature and flowering time in wheat—Assisting breeder to keep pace with climate change. Glob. Chang. Biol. 22:921–933. doi:10.1111/gcb.13118

Zhuang, Y., A. Gala, and Y. Yen. 2013. Identification of functional genic components of major *Fusarium* head blight resistance quantitative trait loci in wheat cultivar Sumai 3. Mol. Plant-Microbe Interact. 26:442–450. doi:10.1094/MPMI-10-12-0235-R

VITA

Education:

M.S., Plant Breeding, University of Pelotas, Brazil, 2013

B.S., Agronomy, University of Pelotas, Brazil, 2011

Publications:

Referred Journals:

Tessmann, E.W., D.A. Van Sanford. Associations between morphological and FHB traits in a soft red winter wheat population. *Under consideration – Euphytica.*

Tessmann, E.W., D. Yanhong, and D.A. Van Sanford. 2019. GWAS for Fusarium head blight traits in a soft red winter wheat mapping panel. Crop Science. *Accepted for publication.*

Tessmann, E.W., and D. Van Sanford. 2018. GWAS for Fusarium head blight related traits in winter wheat (*Triticum aestivum* L.) in an artificially warmed treatment. Agronomy 8:68. doi:10.3390/agronomy8050068

Nornberg, R., J.A.G. Silva, H.S. Luche, **E.W. Tessmann**, S.A.F. Kavalco, C.M. Zimmer, D. Baretta, L.C. Maia, and A.C. Oliveira. 2015. Tolerance to preharvest sprouting and yield of wheat genotypes from different breeding programs. Pesquisa Agropecuaria Brasileira (Online) 698–706. doi: 10.7213/academica.12.03.AO03

Kavalco, S.A.F., R.G. Figueiredo, E.L. Groli, C.M. Zimmer, D. Baretta, **E.W. Tessmann**, A. Magalhaes-Junior, and A. Costa de Oliveira. 2014. Pathway analysis in wheat genotypes under waterlogging stress. Ciencia Agraria 1683–1696. doi: 10.5433/1679-0359.2014v35n4p1683

Silveira, G., E. Moliterno, G. Ribeira, P.M.A. Costa, L.G. Woyann, **E.W. Tessmann**, A.C. Oliveira, and C.D. Cruz. 2014. Increasing genetic variability in black out using gamma irradiation. Genetics and Molecular Research 10332–10340. doi: 10.4238/2014.December.4.28

Crestani, M., S.F.S. Silveira, **E.W. Tessmann**, I. Mezzalira, H.S. Luche, J.A.G. Silva, L.C. Gutkoski, F.I.F. Carvalho, and A. Costa de Olivera. 2012. Combining ability for grain chemistry quality traits in a white oat diallelic cross. Euphytica (Wageningen) 139–156. doi: 10.1007/s10681-012-0641-0

Crestani, M., J.A. Gonzalez da Silva, **E.W. Tessmann**, I. Mezzalira, A. Costa de Oliveira, and F.I.F. Carvalho. 2011. A proposal for aluminum tolerance selection in white oat under hydroponic conditions. Journal of Crop Science and Biotechnology (Seoul) 71–77. doi: 10.1007/s12892-010-0090-z

Ribeira, G., G. Silveira, M. Crestani, F.I.F. Carvalho, A. Costa de Oliveira, D. Baretta, L.G. Woyann, and **E.W. Tessmann**. 2011. Response of white oat (*Avena sativa* L.) genotypes to manganese under hydroponic culture. R. Bras. Agrociencia 57–66. doi: 10.1007/s11104-011-0937-1

Crestani, M., J.A.G. Silva, **E.W. Tessmann**, S.F.F. Silveira, R. Nornberg, G. Ribeira, F.I.F. Carvalho, and A. Costa de Oliveira. 2011. Protocols for aluminum tolerance in white oat under hydroponic cultivation. R. Bras. Agrociencia 311–320. Doi: 10.1080/00103624.2011.539083

Abstracts and Proceedings (First author):

Tessmann, E.W., and D.A. Van Sanford. 2018. GWAS for Fusarium head blight traits in the Elite Eastern Wheat Mapping panel. 2018 Fusarium Head Blight Forum. St. Luis, Missouri.

Tessmann, E.W., and D.A. Van Sanford. 2017. GWAS for scab traits in the Elite Eastern Wheat Mapping panel in an artificially warmed environment. 2017 National Fusarium Head Blight Forum. Milwaukee, Wisconsin.

Tessmann, E.W., and D.A. Van Sanford. 2017. GWAS of morphological and scab resistance traits in the Elite Eastern Wheat Mapping panel. American Society of Agronomy, Crop Science of America and Soil Science Society of America joint Annual Meeting. Tampa, Florida.

Tessmann, E.W., A. Clark, D.A. Van Sanford. 2016. Morphological and FHB trait variation in the Elite Eastern Mapping panel. 2016 National Fusarium Head Blight Forum. St. Luois, Missouri.

Tessmann, E.W., L.G. Woyann, G. Silveira, R. Danielowski, E. Moliterno, J. Silva, L.C. Maia, and A. Costa de Oliveira. 2013. Performance of black oat (*Avena strigose* Schreb.) population to different doses of EMS mutagenic. XXXIII Committee Meeting of the Brazilian Oat Research. Pelotas, RS, Brazil.

Tessmann, E.W., C.M. Zimmer, E.L. Groli, R.G. Figueiredo, R.L. Santos, S.A.F. Kavalco, L.C. Maia, and A. Costa de Oliveira. 2012. Effect of stress to manganese in phenotypic correlations among characters of white oat. XXXII Committee Meeting of the Brazilian Oat Research. Passo Fundo, RS, Brazil.

Tessmann, E.W., R. Nornberg, H.S. Luche, S.A.F. Kavalco, C.M. Zimmer, E.L. Groli,

L.C. Maia, and A. Costa de Oliveira. 2012. Genetic distance among white oat cultivars (Avena sativa L.) in response to manganese stress. 6th International Crop Science Congress. Bento Gonçalves, RS, Brazil.

Tessmann, E.W., M. Crestani, D. Baretta, C.M. Zimmer, L.G. Woyann, F. Freo, J. Silva, F.I.F. Carvalho, and A. Costa de Oliveira. 2011. Performance of white oat cultivar IAC 7 regard to quality and industrial yield in different cultivation environments. XXXI Committee Meeting of the Brazilian Oat Research Passo Fundo, RS, Brazil.

Tessmann, E.W., D Baretta, M. Crestani, R. Nornberg, I. Mezzalira, H.S. Luche, C.M. Zimmer, R. Paiva, S. Silveira, L.G. Woyann, L.R. Souza, L.F.C. Girardon, S.A.F. Kavalco, T. Hagemann, P.S. Silva, F.F. Cima, F.I. Carvalho, L.C. Maia, and A. Costa de Oliveira. 2010. Brazilian test of white oat cultivars in Pelotas, RS, 2009. In: XXX Committee Meeting of the Brazilian Oat Research. São Carlos, SP, Brazil.

Tessmann, E.W., T. Finatto, T.R. Hagemann, S. Silveira, B.E. Menegussi, D. Baretta, L.C. Maia, and A. Costa de Oliveira. 2010. Microsatellite markers prospection *in silico* for the character preharvest germination based on the metabolism of carotenoids. IV Committee Meeting Brazilian Research Wheat and Triticale. Casacavel, PR, Brazil.

Tessmann, E.W., T. Finatto, L.C. Maia, N. Picault, and A. Costa de Oliveira. 2010. Super gene expression of peroxidases (EC 1.11.1.7) in rice under stress by iron toxicity. In: XIX Undergraduate research meeting, XII Graduate Meeting, Pelotas-RS. 2010.

Tessmann, E.W., M. Crestani, S. Silveira, H.S. Luche, D. Baretta, G. Ribeiro, J. Silva, F.I.F. Carvalho, and A. Costa de Oliveira. 2009. Performance of white oat genotypes (*Avena sativa* L.) submitted to aluminum stress. XVIII Undergraduate research meeting, XI Graduate Meeting. Pelotas, RS, Brazil.

Tessmann, E.W., T. Finatto, I. Mezzalira, R. Nornberg, R. Paiva, P.S. Silva, M. Crestani, F.I.F. Carvalho, and A. Costa de Oliveira. 2009 Behavior of rice genotypes (*Oryza sativa* L.) in response to zinc toxicity in nutrient solution. XVIII Undergraduate research meeting, XI Graduate meeting. Pelotas, RS, Brazil.

Tessmann, E.W., M. Crestani, S. Silveira, L.G. Woyann, D. Baretta, R. Paiva, C. Bervald, F.I.F. Carvalho, and A. Costa de Oliveira. 2009. Phenotypic correlation among seedling characters in selection of oat genotypes tolerance to Al+3. XXIX Committee Meeting Brazilian Oat Research, Porto Alegre, RS, Brazil.

Tessmann, E.W., M. Crestani, S. Silveira, H.S. Luch, D. Baretta, G. Ribeiro, J. Silva, F.I.F. Carvalho, A.C. Oliveria. 2009. Stress by aluminum in white oat genotypes in hydroponic cultivation. XXIX Committee Meeting of the Brazilian Oat Research, Porto Alegre, RS, Brazil.

Tessmann, E.W., M. Crestani, I. Mezzalira, S. Silveira, H.S. Luch, T.R. Hagemann, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Performance of white oat genotypes under different protocols and doses of aluminum. XVII Undergraduate research meeting, X Graduate Meeting, Pelotas, RS, Brazil.

Tessmann, E.W., M. Crestani, G. Silveira, C.M.B. Priebe, L.C. Maia, J. Silva, F.I.F. Carvalho, A. Costa de Oliveira. 2008. Correlations among seedling characters in assessing of white oat genotypes to tolerance for aluminum in hydroponic conditions. XVII Undergraduate research meeting, X Graduate Meeting, Pelotas, RS, Brazil.

Tessmann, E.W., M. Crestani, I. Mezzalira, S. Silveira, G. Ribeiro, J. Silva, F.I.F. Carvalho, A. Costa de Oliveira. 2008. Performance of white oat genotypes under different protocols and doses of aluminum. XVII Undergraduate research meeting, X Graduate Meeting, Pelotas, RS, Brazil.

Abstracts and Proceedings (second author):

Woyann, L., **E.W. Tessmann**, G. Silveira, D. Baretta, L.C. Maia, A. Costa de Oliveria. 2013. Mutagenic agent MMS provides yield increment on M4 populations of black oat. XXXIII Committee Meeting of the Brazilian Oat Research, Pelotas, RS, Brazil.

Thurow, L.B., **E.W. Tessmann**, R. Danielowski, C.M. Zimmer, E.L. Groli, L.C. Maia, A. Costa de Oliveira. 2013. Phenotypic, genotypic and environmental correlations for seedling traits in white oat genotypes under different concentrations of Mn+2. XXXIII Committee Meeting of the Brazilian Oat Research. Pelotas, RS, Brazil.

Kavalco, S.A.F., **E.W. Tessmann**, R.G. Figueiredo, E.L. Groli, C.M. Zimmer, R.L. Santos, R. Nornberg, L.C. Maia, A. Costa de Oliveira. 2012. Diallel analysis in wheat genotypes subjected to flooding stress. 6th International Crop Science Congress. Bento Gonçalves, RS, Brazil.

Kavalco, S.A.F., **E.W. Tessmann**, R.L. Santos, R.G. Figueiredo, C.M. Zimmer, H.S. Luch, L.C. Maia, A. Costa de Oliveira. 2012. Path analysis in genotypes of oat submitted to stress for manganese. XXXII Committee Meeting Brazilian Oat Research. Passo Fundo, RS, Brazil.

Crestani, M., **E.W. Tessmann**, S. Silveira, H.S. Luch, R. Nornberg, M. Cerioli, S.A.F. Kavalco, J. Silva, L.C. Gutkoski, A. Costa de Oliveira, F.I.F. Carvalho. 2012. Relationship among F1 and F2 generations of white oat for chemical quality of grains. XXXII Committee Meeting of the Brazilian Oat Research. Passo Fundo,RS, Brazil.

Zimmer, C.M., **E.W. Tessmann**, E.L. Groli, R. Danielowski, M. Olivo, R. Nornberg, L.C. Maia, A. Costa de Oliveira. 2012. Response of white oat cultivars to root length

character under Mn^{2+} stress in hydroponic cultivation. XXXII Committee Meeting of the Brazilian Oat Research. Passo Fundo, RS, Brazil.

Mezzalira, I., **E.W. Tessmann**, S. Silveira, T. Finatto, M. Crestani, L.C. Maia, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Response of rice genotypes to different zinc concentrations in nutrient solution. XVIII Undergraduate research meeting, XI Graduate Meeting. Pelotas, RS, Brazil.

Abstracts and Proceedings (others):

Thurow, L.B., V. Oliveria, G.G. Conte, **E.W. Tessmann**, R. Danielowski, T.P. Viana, A. Costa de Oliveria. 2014. Estimation of the genetic distance for wheat characters related to yield components. VIII Committee Meeting of the Brazilian wheat and triticale Research. Canela, RS, Brazil.

Nornberg, R., H.S. Luche, **E.W. Tessmann**, S.A.F. Kavalco, C.M. Zimmer, E.L. Groli, R.G. Figueiredo, L.C. Maia, A. Costa de Oliveira. 2013. Variability for resistance to pre-harvest sprouting in wheat populations. 7° Plant Breeding Brazilian Meeting. Uberlandia, MG, Brazil.

Baretta, D., S.A.F. Kavalco, C.M. Zimmer, **E.W. Tessmann**, C. Busanello, R. Nornberg, E. Moliterno, J. Silva, L.C. Maia, Costa de Oliveira. 2013. Path analysis using phenotypic values for dry mass production for white oat genotypes. XXXIII Committee Meeting of the Brazilian Oat Research. Pelotas, RS, Brazil.

Busanelo, C., D. Baretta, **E.W. Tessmann**, M. Nardino, D.N. Follmann, V.Q. Souza, L.C. Maia, A. Costa de Oliveira. 2013. Development of white oat under the scenario of climate change. XXXIII Committee Meeting of the Brazilian Oat Research. Pelotas, RS, Brazil.

Conte, G.G., M.Z. Polesello, V. Oliveira, **E.W. Tessmann**, A. Costa de Oliveira, L.C. Maia. 2013. Multivariate and correlation analysis of wheat (*Triticum aestivum*) genotypes in the Pelotas region. XXXIII Committee Meeting of the Brazilian Oat Research. Pelotas, RS, Brazil.

Nornberg, R., H.S. Luch, **E.W. Tessmann**, M. Olivo, C.M. Zimmer, R.G. Figueiredo, L.C. Maia, A. Costa de Oliveira. Estimate of heterosis and inbreeding depression for character Falling Number wet in wheat. 6th International Crop Science Congress. Bento Gonçalves, RS, Brazil.

Woyann, L.G., T. Finatto, **E.W. Tessmann**, L.C. Maia, A. Costa de Oliveira. 2012. Regulation of receptor-like kinase (RLK) gene family responsive to iron excess in rice. 6th International Crop Science Congress. Bento Gonçalves, RS, Brazil.

Olivo, M., D. Baretta, **E.W. Tessmann**, R. Danielowski, A. Magalhaes-Junior, A. Costa

de Oliveira, L.C. Maia. 2012. Response of rice cultivars subjected to germination in presence of iron. XXIII Congress Pan-American of Seeds. Santa Cruz de La Sierra, Bolivia.

Santos, R.L., H.S. Luch, **E.W. Tessmann**, R. Nornberg, S. Silveira, A. Cardoso, L.C. Maia, A. Costa de Oliveira. 2012. Agronomic performance of white oat lines in different years in the region of Pelotas/RS. XXXII Committee Meeting of the Brazilian Oat Research. Passo Fundo, RS, Brazil.

Luch, H.S., M. Olivo, R. Nornberg, S.A.F. Kavalco, **E.W. Tessmann**, R.L. Santos, J. Silva, L.C. Maia, A. Costa de Oliveira. 2012.Adaptive traits in oat breeding in Brazil. 6th International Crop Science Congress. Bento Gonçalves, RS, Brazil.

Groli, E.L., C.M. Zimmer, D. Baretta, R.L. Santos, L.G. Woyann, H.C.F. Almeida, **E.W. Tessmann**, R. Nornberg, L.C. Maia, A. Costa de Oliveria. 2012. Reginal test of white oat line ERLA, Pelotas/RS – 2011. XXXII Committee Meeting of the Brazilian Oat Research. Passo Fundo, RS, Brazil.

Kavalco, S.A.F., H.S. Luch, E.L. Souza, E.L. Groli, F.F. Cima, P.S. Silva, S.I. Costa, **E.W. Tessmann**, L.C. Maia, A. Costa de Oliveira. 2011. Analysis of comparison means to yield in foreign genotypes of white oat conducted in three years in Capão do Leão/RS. XXXII Committee Meeting of the Brazilian Oat Research. Passo Fundo, RS, Brazil.

Nornberg, R., H.S. Luch, S.A.F. Kavalco, D. Baretta, L.F.C. Girardon, S. Silveira, **E.W. Tessmann**, C.M. Zimmer, A. Costa de Oliveira. 2011. Adaptability and stability evaluation in white oat genotypes conducted over three years in Pelotas. XXXII Committee Meeting of the Brazilian Oat Research. Passo Fundo, RS, Brazil.

Crestani, M., D. Baretta, **E.W. Tessmann**, R. Paiva, S.A.F. Kavalco, J. Silva, J.F. Sartori, F.I.F. Carvalho, A. Costa de Oliveira. 2011. Genetic distance among white oat cultivars based on agronomic traits and industrial quality. XXXI Committee Meeting of the Brazilian Oat Research. Passo Fundo, RS, Brazil.

Girardon, L.F.C., C.M. Zimmer, E.L. Groli, **E.W. Tessmann**, F. Freo, S.I. Costa, M. Crestani, H.S. Luch, S.A.F. Kavalco, L.C. Maia, A. Costa de Oliveira. 2011. Brazilian test of white oat lines – EBLA, Pelotas/RS 2010. XXXI Committee Meeting of the Brazilian Oat Research. Passo Fundo, RS, Brazil.

Kavalco, S.A.F., H.S. Luch, R. Nornberg, S. Silveira, F. Freo, Woyann, L.G., E.L. Groli, E.L. Souza, **E.W. Tessmann**, L.C. Maia, A. Costa de Oliveira. 2011. Characterization and study of the effect of submersion stress in wheat genotypes aiming the tolerance. 6º Brazilian Congress of Plant Breeding. Búzios, RJ, Brazil.

Zimmer, C.M., M. Crestani, **E.W. Tessmann**, H.S. Luch, M. Cerioli, S. Silveira, L.G. Woyann, D. Baretta, I. Mezzalira, R. Nornberg, R. Paiva, L.C. Maia, F.I.F.

Carvalho, A. Costa de Oliveira. 2010. Correlation between interested agronomic traits in white oat (*Avena sativa* L.) and prediction of superior crosses. In: XXX Committee Meeting of the Brazilian Oat Research. São Carlos, SP, Brazil.

Silveira, S., M. Crestani, **E.W. Tessmann**, M. Wachholz, A. Costa de Oliveira. 2010. Resumption of root growth in white oat genotypes under different protocols and aluminum doses. XIX Undergraduate research meeting, XII Graduate Meeting. Pelotas, RS, Brazil.

Paiva, R. C.M. Zimmer, D. Baretta, **E.W. Tessmann**, M. Crestani, I. Mezzalira, H.S. Luch, M. Cerioli, R. Nornberg, S. Silveira, L.G. Woyann, T. Finatto, L.R. Souza, L.F.C. Girardon, S.A.F. Kavalco, T.R. Hagemann, B.E. Menegussi, L.C. Maia, F.I.F. Carvalho, A. Costa de Oliveira. 2010. Brazilian test of white oat lines in Pelotas – RS, 2009. XXX Committee Meeting of the Brazilian Oat Research. São Carlos, SP, Brazil.

Girardon, L.F.C., R. Paiva, S. Silveira, **E.W. Tessmann**, D. Baretta, M. Crestani, I. Mezzalira, H.S. Luch, C.M. Zimmer, R. Nornberg, M. Cerioli, L.G. Woyann, B.E. Menegussi, F.F. Cima, P.S. Silva, J.P. Silva, L.R. Souza, L.C. Maia, F.I.F. Carvalho, A. Costa de Oliveira. 2010. Regional test of white oat lines – ERLA, Pelotas/RS – 2009. XXX Committee Meeting of the Brazilian Oat Research. São Carlos, SP, Brazil.

Freo, F., S.A.F. Kavalco, F.F. Cima, P.S. Silva, M. Wachholz, **E.W. Tessmann**, L.C. Maia, A. Costa de Oliveira. 2010. Studies of the molecular function associated with genomic regions expressed in wheat (*Triticum aestivum* L.). 19º Undergraduate research meeting, 9º Graduate Meeting and 2º Congress of Extension. Pelotas, RS, Brazil.

Baretta, D., M. Wachholz, C.M. Zimmer, R. Paiva, **E.W. Tessmann**, M. Crestani, F.I.F. Carvalho, A. Costa de Oliveira. Correlation between agronomic traits in wheat (*Triticum aestivum* L.) lines. IV Committee Meeting of the Brazilian Research Wheat and Triticale. Casacavel, PR, Brazil.

Silveira, S., M. Crestani, R. Nornberg, R. Paiva, C.M. Zimmer, D. Baretta, **E.W. Tessmann**, P.S. Silva, J.P. Silva, L.C. Maia, F.I.F. Carvalho, A. Costa de Oliveira. 2010. Correlation among traits in genotypes of white oat subjected to stress by Al^{3+} in different doses and protocols. XXX Committee Meeting of the Brazilian Oat Research. São Carlos, SP, Brazil.

Crestani, M., F.I.F. Carvalho, A. Costa de Oliviera, J. Silva, L.C. Gutkoski, J.F. Sartori, D. Baretta, H.S. Luch, **E.W. Tessmann**, R. Paiva. 2010. Performance of white oat cultivars regard to content of β-glycan in grains in different environments. XXX

Baretta, D., S. Silveira, l.G. Woyann, C.M. Zimmer, R. Paiva, M. Cerioli, **E.W. Tessmann**, H.S. Luch, R. Nornberg, F.F. Cima, M. Crestani, T.R. Hagemann, D.R. Farias,

S.A.F. Kavalco, F.I.F. Carvalho, L.C. Maia, A. Costa de Oliveira. 2010. Preliminary internal test of strains of white oat – PITSO, Pelotas/RS, 2009. XXX Committee Meeting of the Brazilian Oat Research. São Carlos, SP, Brazil.

Silveira, S., M. Crestani, **E.W. Tessmann**, I. Mezzalira, H.S. Luche, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Assessment of avenacor in white oat cultivars conducted in Pelotas/RS in 2007 and 2008. XVIII Undergraduate research meeting, VIII Graduate Meeting. Pelotas, RS, Brazil.

Woyann, L.G., M. Cerioli, **E.W. Tessmann**, R. Nornberg, R. Paiva, G. Ribeiro, G. Silveira, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Brazilian test of white oat lines – EBLA, Pelotas/RS – 2008. XXIX Committee Meeting of the Brazilian Oat Research. Porto Alegre, RS, Brazil.

Zimmer, C.M., C.C. Mistura, **E.W. Tessmann**, I. Mezzalira, A. Magalhaes-Junior, A. Costa de Oliveira, F.I.F. Carvalho. 2009. Identification of genetic variability in rice (*Oryza sativa* L.) regard to root system. XVIII Undergraduate research meeting, XI Graduate Meeting. Pelotas, RS, Brazil.

Silva, J.P., S. Silveira, **E.W. Tessmann**, I. Mezzalira, L.C. Maia, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Occurrence of microsatellites in the rice genome. XVIII Undergraduate research meeting, XI Graduate Meeting. Pelotas, RS, Brazil.

Mezzalira, I., G. Silveira, **E.W. Tessmann**, H.S. Luche, S. Silveira, F.F. Cima, E. Moliterno, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Yield and seed dormancy in of black oat (*Avena strigosa* Schereb.) hybrid populations and their parents. XXIX Committee Meeting of the Brazilian Oat Research. Porto Alegre, RS, Brazil.

Baretta, D., H.S. Luche, R. Nornberg, **E.W. Tessmann**, S. Silveira, M. Crestani, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Phenotypic correlation in characters related to yield in white oat genotypes conducted in different sowing systems. XXIX Committee Meeting of the Brazilian Oat Research. Porto Alegre, RS, Brazil.

Luche, H.S., R. Nornberg, D. Baretta, **E.W. Tessmann**, M. Crestani, G. Ribeiro, A. Costa de Oliveira, F.I.F. Carvalho. 2009. Estimation of genetic dissimilarity and phenotypic correlation in white oat genotypes. XVIII Undergraduate research meeting, XI Graduate Meeting. Pelotas, RS, Brazil.

Crestani, M. S. Silveira, I. Mezzalira, **E.W. Tessmann**, G. Ribeira, J. Silva, L.C. Gutkoski, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Relation of yield and industrial components traits and chemical quality in grains of white oat. XXIX Committee Meeting of the Brazilian Oat Research. Porto Alegre, RS, Brazil.

Nornberg, R., D. Baretta, M. Cerioli, **E.W. Tessmann**, M. Crestani, G. Ribeiro, T.M. Souza, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Response of white oat

genotypes (*Avena sativa* L.) to manganese in hydroponic cultivation. XXIX Committee Meeting of the Brazilian Oat Research. Porto Alegre, RS, Brazil.

Silveira, S., M. Crestani, I. Mezzalira, **E.W. Tessmann**, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Content of grain total protein of white oat cultivars. XVIII Undergraduate research meeting, VIII Graduate Meeting and Extension Congress of Catholic University of Pelotas (UCPel). Pelotas, RS, Brazil.

Mezzalira, I., M. Crestani, S. Silveira, **E.W. Tessmann**, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Performance of white oat (Avena sativa L.) genotypes submitted to aluminum toxicity in hydroponic. XVIII Undergraduate research meeting, VIII Graduate Meeting and Congress Extension of Catholic University of Pelotas (UCPel). Pelotas,RS, Brazil.

Luche, H.S., D.A.M. Shmidt, R. Nornberg, **E.W. Tessmann**, G. Ribeiro, A. Costa de Oliveira, F.I.F. Carvalho. 2009. Studies of heterosis in diallel crosses of wheat. III Committee Meeting Brazilian Research Wheat and Triticale. Veranópolis, RS, Brazil.

Lopes, M.R.S., L. Amarante, V.C. Chaves, L.A. Luz, **E.W. Tessmann**, D.H. Carvalho. 2009. Activity of nitrate reductase and glutamine synthetase *in Brugmansia suaveolens* cultivated under different nitrogen sources. XII Brazilian Congress of Plant Physiology. Fortaleza, CE, Brazil.

Silveira, S., L.C. Maia, M. Crestani, J.P. Silva, **E.W. Tessmann**, R. Nornberg, H.S. Luche, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Studies *in silico* of conservation of gene between oat and rice genomes. XXIX Committee Meeting of the Brazilian Oat Research. Porto Alegre, RS, Brazil.

Silveira, S., M. Crestani, D. Baretta, I. Mezzalira, **E.W. Tessmann**, C. Bervald, G. Silveira, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Factors determining the efficiency of artificial hybridizations between white oat genotypes. XXIX Committee Meeting of the Brazilian Oat Research. Porto Alegre, RS, Brazil.

Farias, D.R., L.C. Maia, R.J. Ahlert, C. Bervald, I. Mezzalira, **E.W. Tessmann**, F.I.F. Carvalho, A. Costa de Oliveira. 2009. Identification of similarity between sequences EST of wheat (*Triticum aestivum* L.) in EST database of oat (*Avena sativa* L.). XXIX Committee Meeting of the Brazilian Oat Research. Porto Alegre, RS, Brazil.

Mezzalira, I., N. Marini, D.R. Farias, **E.W. Tessmann**, S. Silveira, T.M. Souza, F.I.F. Carvalho, A. Costa de Oliveira. 2008. Characterization of oat cultivars (*Avena sativa* L.) tolerance to flooding and waterlogging stress. XVII Undergraduate research meeting, X Graduate Meeting. Pelotas, RS, Brazil.

Cerioli, M., R. Nornberg, M.T. Stumpf, **E.W. Tessmann**, M. Crestani, G. Ribeiro, F.I.F. Carvalho, A. Costa de Oliveira. 2008. Estimation of heterosis in diallel crosses in hexaploid wheat. XVII Undergraduate research meeting, X Graduate Meeting. Pelotas, RS, Brazil.

Luche, H.S., L.G. Woyann, M. Cerioli, **E.W. Tessmann**, D.A.M. Shmidt, L.C. Gutkoski, F.I.F. Carvalho, A. Costa de Oliveira. 2008. Industrial quality of wheat genotypes in response to nitrogen conducted in different environments. XVII Undergraduate research meeting, X Graduate Meeting. Pelotas, RS, Brazil.

Crestani, M., F. Hawerroth, J. Silva, F.I.F. Carvalho, A. Costa de Oliveira, D.A.M. Shmidt, L.G. Woyann, R. Nornberg, M.T. Stumpf, **E.W. Tessmann**. 2008. Correlation between climatic variables and agronomic traits in white oat genotypes conducted in Pelotas-RS. XXVIII Committee Meeting of the Brazilian Oat Research. Pelotas, RS, Brazil.

Souza, T.M., D. Colares, D. Baretta, S. Silveira, I. Mezzalira, **E.W. Tessmann**, A.M. Almeida, F.I.F. Carvalho, A. Costa de Oliveira. 2008. Effect of Fe^{2} on growth parameters of rice genotypes under field conditions. XVII Undergraduate research meeting, X Graduate Meeting. Pelotas, RS, Brazil.

Mezzalira, I., A.F. Martins-Dionello, D.R. Farias, M.M. Kopp, **E.W. Tessmann**, S. Silveira, T.M. Souza, A. Magalhaes-Junior, F.I.F. Carvalho, A. Costa de Oliveira. 2008. Genetic variability in families of rice mutants regard to agronomic importance traits. XVII Undergraduate research meeting, X Graduate Meeting. Pelotas, RS, Brazil.

Silveira, S., J.S. Castelo Branco, M. Fagundes, T. Medeiros, **E.W. Tessmann**, A. Costa de Oliveira. 2008. Cloning of genes ADH1 in rice and wheat genotypes. XVII Undergraduate research meeting, X Graduate Meeting. Pelotas, RS, Brazil.

Elisane Weber Tessmann